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RESPIRATORY CARE

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- Pressure-Control Ventilation with a Puritan-Bennett 7200a Ventilator: Application of an Algorithm and Results in 14 Patients
- Computers in Pulmonary Medicine: Ten Years Ago, Today, and Tomorrow
  - Computerized Management of Respiratory Care
  - Quality Assurance for Respiratory Care Services: A Computer-Assisted Program
  - Using Belief Networks To Interpret Qualitative Data in the ICU
  - Microprocessor-Based Mechanical Ventilation
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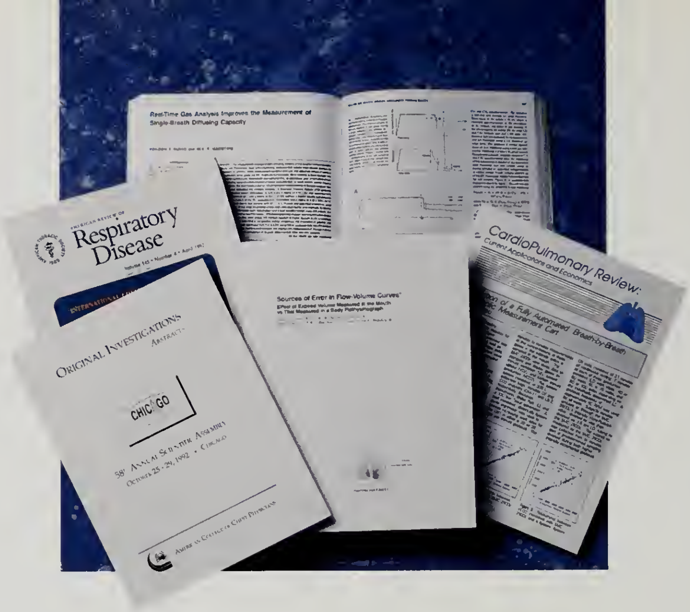
- Classic Reprints—Retrospectroscope 1975: Pulmonary Diffusing Capacity for Carbon Monoxide (D_{LCO})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[1] Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen in hypoxemic chronic obstructive lung disease: a clinical trial. *Ann Intern Med.* 1980; 93:391-8.

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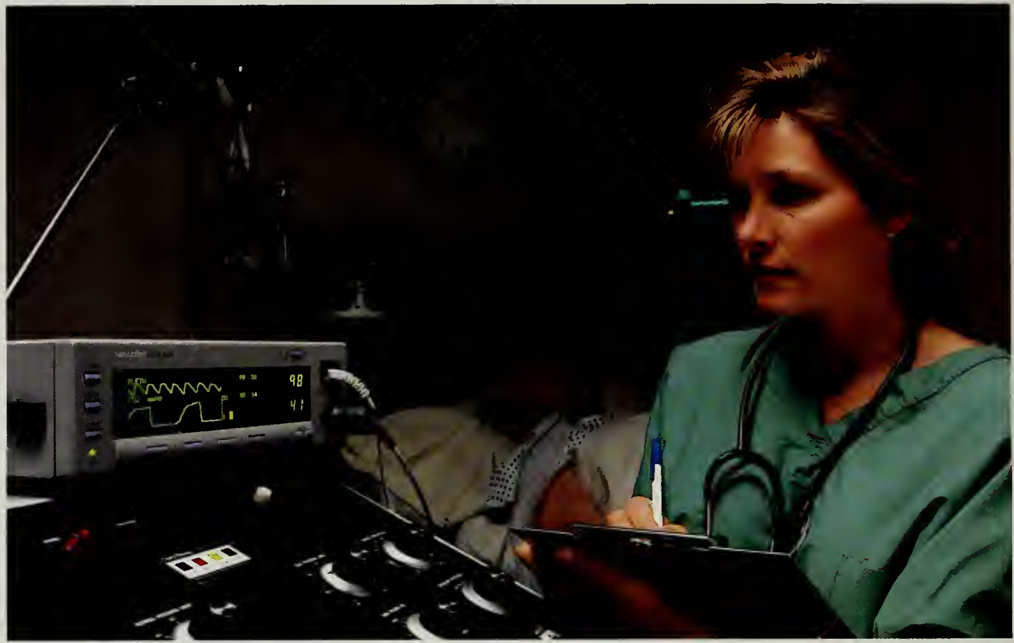

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The Increasing Problem of Asthma in the United States (editorial)—PJ Gergen, KB Weiss. Am Rev Respir Dis 1992;146:1823.

An RSVP to RSV (Respiratory Syncytial Virus): Declining Mortality Rates (editorial)—MM Pollack. Crit Care Med 1992;20(10):1375. (Pertains to Moler et al paper abstracted on Page 20.)

Reduced Fertility among Women Employed as Dental Assistants Exposed to High Levels of Nitrous Oxide—AS Rowland, DD Baird, CR Weinberg, DL Shore, CM Shy, AJ Wilcox. N Engl J Med 1992;327: 993.

BACKGROUND: Fertility is reduced in female rats exposed to levels of nitrous oxide similar to those found in some dental offices. Epidemiologic studies have suggested an association between exposure to mixed anesthetic gases and impaired fertility. We investigated the effects of occupational exposure to nitrous oxide on the fertility of female dental assistants. METHODS: Screening questionnaires were mailed to 7,000 female dental assistants, ages 18 to 39, registered by the California Department of Consumer Affairs. 69% responded. 459 women were determined to be eligible, having become pregnant during the previous 4 years for reasons unrelated to the failure of birth control, and 91% of these women completed telephone interviews. Detailed information was collected on exposure to nitrous oxide and fertility (measured by the number of menstrual cycles without contraception that the women required to become pregnant). RESULTS: After controlling for covariates, we found that women exposed to high levels of nitrous oxide were significantly less fertile than women who were unexposed or exposed to lower levels of nitrous oxide. The effect was evident only in the 19 women with 5 or more hours of exposure per week. These women were only 41% (95% confidence interval, 23-74%; $p < 0.003$) as likely as unexposed women to conceive during each menstrual cycle. CONCLUSIONS: Occupational exposure to high levels of nitrous oxide may adversely affect a woman's ability to become pregnant.

Clinical Determinants of the Racial Disparity in Very Low Birth Weight—A Kempe, PH Wise, SE Barkan, WM Sappenfield, B Sachs, SL Gortmaker, et al. N Engl J Med 1992;327:969.

BACKGROUND: Although the risk of very low birth weight (< 1,500 g) is more than twice as high among blacks as among whites in the United States, the clinical conditions associated with this disparity remain poorly explored. METHODS & RESULTS: We reviewed the medical records of over 98% of all infants weighing 500-1,499 g who were born in Boston during the period 1980–1985 (687 infants), in St Louis in 1985 and 1986 (397 infants), and in two health districts in Mississippi in 1984 and 1985 (215 infants). The medical records of the infants' mothers were also reviewed. These data were linked to birth-certificate files. During the study periods, there were 49,196 live births in Boston, 16,232 in St Louis, and 16,332 in the Mississippi districts. The relative risk of very low birth weight among black infants as compared with white infants ranged from 2.3-3.2 in the three areas. The higher proportion of black infants with very low birth weights was related to an elevated risk in their mothers of major conditions associated with very low birth weight, primarily chorioamnionitis or premature rupture of the amniotic membrane (associated with 38.0% of the excess proportion of black infants with very low birth weights [95% confidence interval 31.3-45.4%]);

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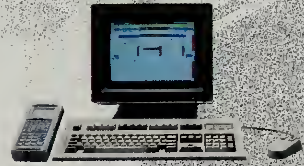

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idiopathic preterm labor (20.9% of the excess [95% confidence interval 16.0-26.4%]); hypertensive disorders (12.3% [95% confidence interval 8.6-16.6%]); and hemorrhage (9.8% [95% confidence interval 5.5-13.5%]). CONCLUSIONS: The higher proportion of black infants with very low birth weights is associated with a greater frequency of all major maternal conditions precipitating delivery among black women. Reductions in the disparity in birth weight between blacks and whites are not likely to result from any single clinical intervention but, rather, from comprehensive preventive strategies.

A Prospective Investigation of Pulmonary Embolism in Women and Men—DA Quinn, BT Thompson, ML Terrin, JH Thrall, CA Athanasoulis, KA McKusick, PD Stein, CA Hales. JAMA 1992;268:1689.

OBJECTIVE: The aim of this study was to compare, in women and men suspected of pulmonary embolism, the frequency, risk factors, diagnosis, and presentation of pulmonary embolism as well as the accuracy of the ventilation/perfusion scan (V/Q scan) as a diagnostic tool. DESIGN: Data were collected during a prospective study (the Prospective Investigation of Pulmonary Embolism Diagnosis) to establish the accuracy of the V/Q scan compared with pulmonary angiograms. SETTING: 6 tertiary medical centers in Massachusetts, Michigan, Connecticut, Pennsylvania, and North Carolina. PARTICIPANTS: Patients suspected of pulmonary embolism for whom a request was made for a V/Q scan or pulmonary angiogram (496 women and 406 men). RESULTS: Women 50 years old and under had a decreased frequency of pulmonary embolism compared with men of that age (16% vs 32%), but there was no difference in patients over 50 years old (Breslow-Day test, $p < 0.01$). Risk factors for pulmonary embolism, the usefulness of the V/Q scan, and 1-year mortality were not different for women and men. Estrogen use in women was not associated with an increased frequency of pulmonary embolism, except in women using oral contraceptives who had undergone surgery within 3 months; 4 of 5 (80%) had emboli compared with 4 of 28 (14%) age-matched surgical patients not using estrogens ($p < 0.01$). CONCLUSION: Women 50 years old and under (even young women using oral contraceptives) who were suspected of having pulmonary emboli and were enrolled in the Prospective Investigation of Pulmonary Embolism Diagnosis study had a smaller frequency of pulmonary embolism than men of that age. The risk factors for pulmonary embolism were the same for women and men, except that women using oral contraceptives had an increased risk of pulmonary embolism following surgery. Although the V/Q scan was a useful tool in the preliminary evaluation for pulmonary embolism in these women, a pulmonary angiogram was often needed for accurate diagnosis.

Active and Passive Smoking and Pathological Indicators of Lung Cancer Risk in an Autopsy Study—D Trichopoulos, F Mollo, L Tomatis, E Agapitos, L Delsedime, X Zavitsanos, et al. JAMA 1992;268:1697.

OBJECTIVE: The association between involuntary smoking and lung cancer has been supported by most epidemiologic studies, but a number of authors and interest groups claim that the possibility of bias has not been excluded. Few autopsy-based studies have explored the role of active smoking and other exposures in lung carcinogenesis, and none has been previously done to examine the role of passive smoking. We have undertaken such an autopsy-based study in Athens, Greece. DESIGN: Lung specimens were taken at autopsy from 400 persons 35 years of age or older, of both genders, who had died within 4 hours from a cause other than respiratory or cancer in Athens or the surrounding area. For each person at least 7 tissue blocks were taken from the main and lobar bronchi and at least 5 blocks from the parenchyma, including an average of about 20 smaller cartilaginous bronchi and membranous bronchioles. The specimens were examined without knowledge of the exposures of the particular subject, in Turin, Italy. For 283 (71%) of the subjects the preservation of the bronchial epithelium was satisfactory for pathological examination, and for 206 among them (73%) an interview could be arranged with their next of kin, focusing on smoking habits of the deceased and their spouses, as well as other variables. The interviewers were not aware of the results of the pathological examinations. MAIN OUTCOME MEASURE: Specimens were examined for basal cell hyperplasia, squamous cell metaplasia, cell atypia, and (in membranous bronchioles and bronchiolo-alveolar airways) mucous cell metaplasia, ie, pathological entities that may be lung cancer risk indicators or epithelial, possibly precancerous, lesions (EPPL). The gland and wall thicknesses were also measured and their ratio calculated (Reid Index). RESULTS: In comparison with nonsmokers, EPPL values were significantly higher among current smokers and higher, but not significantly so, among former smokers. Furthermore, EPPL values were significantly higher among deceased nonsmoking women married to smokers rather than to nonsmokers. In this set of data neither occupation nor residence was associated with EPPL, but this could be due to the poor correlation of residential history with exposure

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Prevention
In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth.

Rescue
To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age.

CONTRAINDICATIONS
None known.

WARNINGS
SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS
General
Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility
Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS
The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	**SURVANTA (%)**	**Control (%)**	***P*-Value[a]**
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	<0.001
Pulmonary interstitial emphysema	20.2	38.4	<0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a] P-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, *P*=0.001; and 48.8% *v* 34.2%, *P*=0.047, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardio-respiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration

Follow-Up Evaluations
To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies
Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies
Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE
Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

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SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0.9% sodium chloride solution. The color is off-white to light brown.

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to air pollution and the lack of adequate standardization of contemporary Greek occupations. The Reid Index was higher among smokers and former smokers in comparison with nonsmokers, among subjects with mainly urban residence in comparison with those with mainly rural residence, and among nonsmoking women married to smokers in comparison with those married to nonsmokers, but none of these differences was statistically significant. CONCLUSION: These results provide support to the body of evidence linking passive smoking to lung cancer, even though they are based on a study methodologically different from those that have previously examined this association.

Hospital Characteristics and Quality of Care—EB Keeler, LV Rubenstein, KL Kahn, D Draper, ER Harrison, MJ McGinty, et al. JAMA 1992;268:1709.

OBJECTIVE: To compare quality of care measured by explicit criteria, implicit review, and sickness-adjusted outcomes at different types of hospitals. DESIGN: Further analysis of data retrospectively abstracted from medical records to evaluate the effects of prospective payment on quality of care for hospitalized Medicare patients. SETTING: Hospitals in 5 states were sampled to represent the national Medicare admissions along many dimensions. PATIENTS: A total of 14,008 elderly patients with 1 of the following 5 diseases: congestive heart failure, acute myocardial infarction, pneumonia, stroke, or hip fracture. These patients were randomly sampled from those with these diseases in 297 hospitals in 2 time periods, 1981 to 1982 and 1985 to 1986. OUTCOME MEASURES: Explicit criteria, implicit review, and mortality within 30 days of admission adjusted for sickness at admission. RESULTS: Quality of care ratings for hospital types are similar using explicit criteria, implicit review, and outcomes adjusted for sickness at admission. Quality differences between types of hospitals were large, with the lowest group estimated to have four percentage points higher mortality than major teaching hospitals in a cohort of patients with average mortality of 16%. Quality varies from state to state, but teaching, larger, and more urban hospitals have better quality in general than nonteaching, small, and rural hospitals. Hospital quality persists over time, but small nonteaching hospitals narrowed the gap with better quality hospitals between 1981 and 1986. CONCLUSIONS: The different measures led to consistent and plausible relationships between quality and hospital characteristics. Thus, valid information about hospital quality can be obtained. We need to develop ways to use such information to improve care.

Diagnosis, Treatment, and Prevention of Pulmonary Embolism: Report of the WHO/International Society and Federation of Cardiology Task Force—SZ Goldhaber, M Morpurgo for the WHO/ISFC Task Force on Pulmonary Embolism. JAMA 1992;268:1727.

To assess the state of the art of venous thrombosis and pulmonary embolism for the medical and other health-related professions, the World Health Organization (WHO) and the International Society and Federation of Cardiology (ISFC) convened a task force in Geneva, Switzerland. Members of the task force prepared position papers and presented brief oral presentations. A report was subsequently prepared by the task force members, who contributed sections in their areas of expertise. Revisions of the report occurred both during the task force meeting itself in Geneva and during the ensuing months. The final report was approved by the WHO-ISFC Task Force on Pulmonary Embolism Steering Committee. More quantitative information is needed on the frequency of venous thrombosis and pulmonary embolism in hospitalized medical patients as well as in outpatients at high risk. Population studies should focus on incidence, survival, and long-term complications in different parts of the world with respect to gender and race. Further educational efforts are needed to increase awareness about venous thrombosis and pulmonary embolism prophylaxis. Finally, research into effective techniques for changing physician practice would be useful.

Apparatus for the Control of Breathing Patterns during Aerosol Inhalation—PR Phipps, I Gonda, SD Anderson. J Aerosol Med 1992;5(3):155.

A computerized breathing circuit for monitoring, recording, and controlling aerosol inhalation patterns is described. A target pattern, using sine functions derived from previous recording of breathing, is displayed on the computer screen. The reproducibility of tidal breathing patterns of 9 normal volunteers was compared using (i) no target, (ii) a metronome and tidal volume (V_T) target, and (iii) the full control provided by this system. Each subject breathed for 2 minutes using each method on separate days. The breath-by-breath variability was found to be similar for all methods. The day-to-day variation was greatest for the 'no control' method with the exception of inspiratory pause (P_i). The inspiratory flowrate (F_i) and P_i had a greater variation using the metronome than the full control. The V_T and breathing frequency were equally well controlled using these two methods. This system provides better control with an easy to follow target

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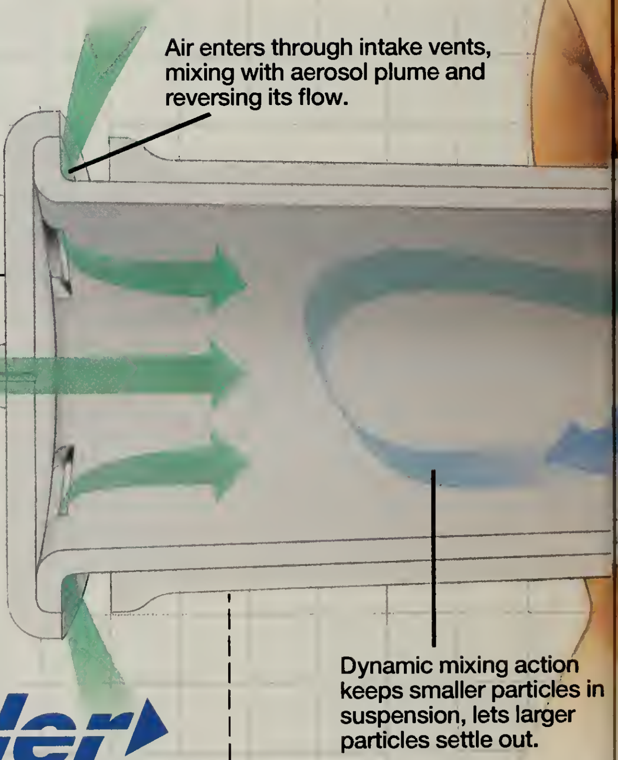
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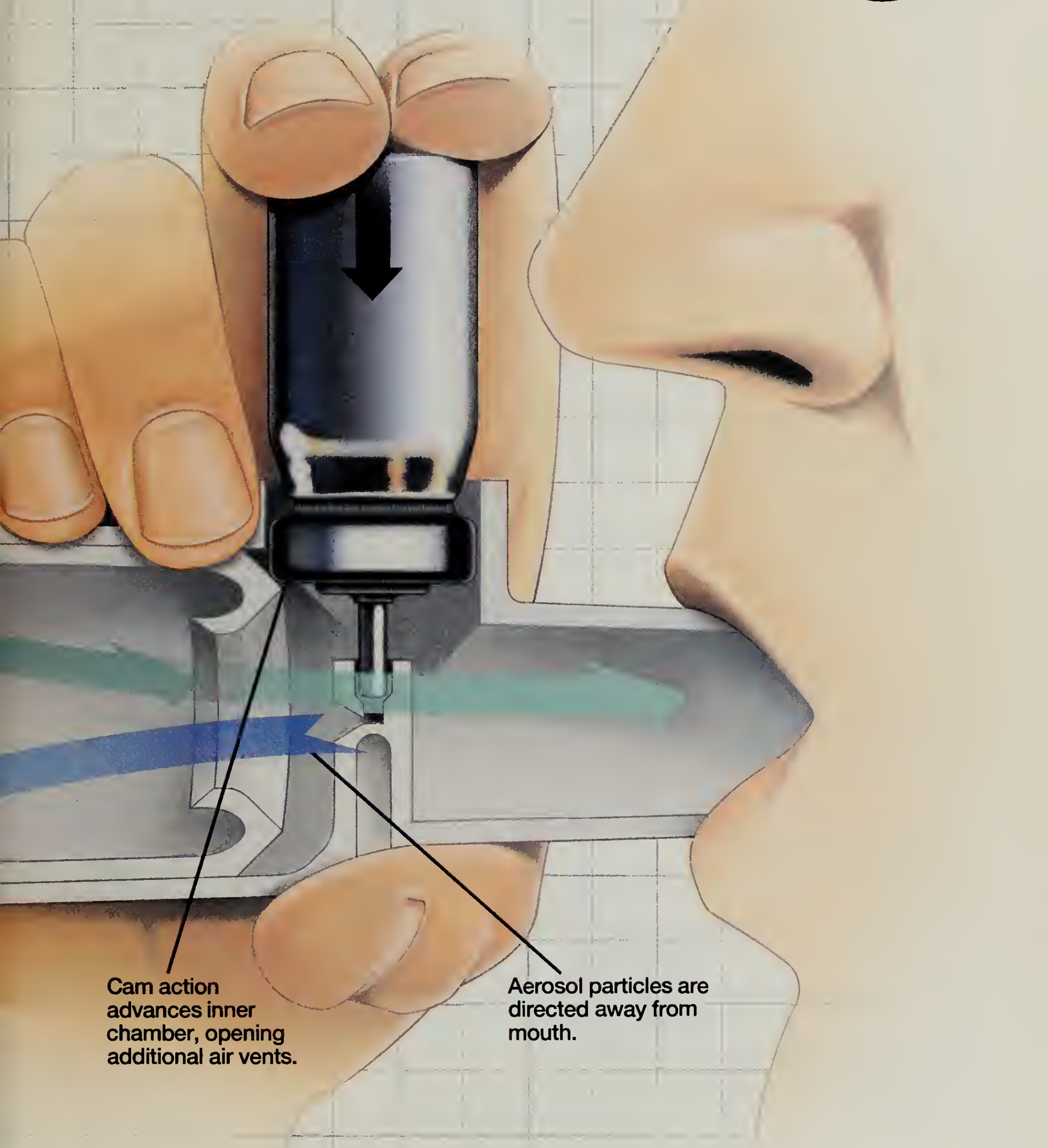
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References: 1-2. Data on file, HealthScan Products Inc. U.S. Patent No. 5,040,527 OA760002-0 8/92

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display of the subject's own, or preset, inhalation pattern.

Transcutaneous and Arterial Blood Gas Monitoring during Acute Asthmatic Symptoms in Older Children—D Holmgren, R Sixt. Pediatr Pulmonol 1992;14:80.

The relationship between transcutaneous and arterial blood gases was investigated in 14 children with asthmatic symptoms, aged 7-15 years, before and after the inhalation of salbutamol. The degree of bronchial obstruction was assessed by forced expiratory volume in one second (FEV_1) and maximum expiratory flow when 25% of FVC remained to be expelled (MEF25). On average the transcutaneous P_{O_2} (tcP_{O_2}) was 1.3 kPa (range 2.6-0 kPa) lower and the transcutaneous P_{CO_2} was 0.6 kPa (range 0-1.5 kPa) higher than the corresponding arterial values ($p < 0.01$). The difference between arterial and transcutaneous P_{O_2} was the same over the whole range of values studied (7.5-14 kPa). After the inhalation of salbutamol, the relationship between transcutaneous and arterial blood gases was not significantly changed. Changes in transcutaneous P_{O_2} correlated to changes in MEF25 ($p < 0.05$), indicating a common denominator, probably the conditions in the peripheral airways. We conclude that the close relationship between transcutaneous and arterial blood gases, even after the inhalation of a beta-2 agonist, indicates that the transcutaneous technique can be used for monitoring acute bronchial obstruction and for evaluating the effects of treatment in children of different ages.

Transcutaneous Blood Gas Monitoring during Salbutamol Inhalations in Young Children with Acute Asthmatic Symptoms—D Holmgren, J Bjure, I Engström, R Sixt, G Sten, G Wennergren. Pediatr Pulmonol 1992;14:75.

The effect of salbutamol inhalations on transcutaneous blood gases was investigated in 23 children (aged 11 mo-2.5 yrs) with asthmatic symptoms. After one salbutamol inhalation there was a mean increase in transcutaneous P_{O_2} (tcP_{O_2}) of 0.5 kPa ($p < 0.01$); after a second dose given 30 minutes later, the mean increase was 1.2 kPa ($p < 0.001$). The increase in tcP_{O_2} after only one dose of salbutamol was significantly correlated to age ($p < 0.01$). No such correlation was observed after a second dose. The overall increase in tcP_{O_2} after two salbutamol inhalations showed a negative correlation to the duration of the current symptomatic period ($p < 0.05$). We conclude that salbutamol inhalations have beneficial effects in young children with acute asthmatic symptoms, even below the age of 18 mo, provided that an adequate dose reaches the lung and preferably at an early stage of obstruction.

Adaptive Control of Inspired Oxygen Delivery to the Neonate—VK Bhutani, JC Taube, MJ Antunes, M Delivoria-Papadopoulos. Pediatr Pulmonol 1992;14:110.

Adaptive adjustment of inspired oxygen (F_{IO_2}), based on a desired percent arterial hemoglobin saturation (S_{O_2}) was achieved by on-line bedside control of the oxygen concentration delivered to the neonate. 14 infants with bronchopulmonary dysplasia (BW 860 ± 80 g; GA 26 ± 1 wk; study age 41 ± 8 d) receiving oxygen-air mixtures by hood were studied. The desired range of S_{O_2} from 92 to 96% with a target value of 95% was determined by pulse oximetry and maintained with adjustment of F_{IO_2} using three modes: (1) standard neonatal intensive care protocol with oxygen delivery evaluated at 20 minute intervals; (2) bedside manual control with F_{IO_2} manipulation every 2 to 5 minutes; and (3) adaptive control with on-line adjustment of F_{IO_2} according to a specifically designed adaptive program. Each study period was of 40-minute duration. S_{O_2} values within a steady 94-96% range was achieved for 54% of the time with standard protocol, compared to 69% ($p < 0.01$) with bedside manual control and 81% ($p < 0.01$) with adaptive control. In addition, fluctuations in S_{O_2} values and overshoots were less apparent with adaptive control of oxygen delivery. These data describe adaptive F_{IO_2} control as an efficient alternative technique for achieving a stable desired range of oxygenation in neonates.

Oxygen Desaturation Complicates Feeding in Infants with Bronchopulmonary Dysplasia after Discharge—L Singer, RJ Martin, SW Hawkins, LJ Benson-Szekely, TS Yamashita, WA Carlo. Pediatrics 1992;90:380.

Recurrent episodes of hypoxemia may affect the growth, cardiac function, neurologic outcome, and survival of infants with bronchopulmonary dysplasia (BPD). As oral feeding might stress these infants by compromising pulmonary function even after hospital discharge, we measured oxygen saturation (S_{aO_2}) via pulse oximetry before, during the initial 10 minutes of, and immediately after oral feeding in 11 patients with BPD, 12 very low birth weight infants, and 23 healthy full-term infants. All infants with BPD had been previously discharged from the hospital after weaning from supplemental oxygen. Studies were done at a mean postconceptional age of 43 weeks while the infants were fed at home by one of their parents. Levels of S_{aO_2} for the three groups were comparable before and during feeds. After feeding, the infants with BPD had significantly lower mean levels of S_{aO_2} (84 ± 8% [SD] vs 93 ± 4% and 93 ± 3%, respectively; $p < 0.01$).

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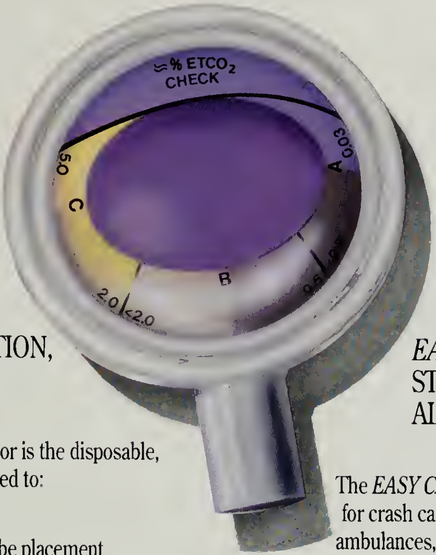

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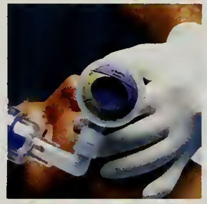

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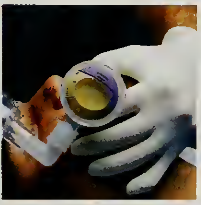

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They also spent more time after feeding with an $S_{aO_2} < 90\%$ (64 ± 34% of time vs 27 ± 33% for the very low birth weight and 22 ± 20% for the term group; $p < 0.01$) and greater time with an $S_{aO_2} < 80\%$ (37 ± 28% vs 4 ± 10% and 4 ± 8%, respectively; $p < 0.01$). Desaturation in infants with BPD was related to larger volume and faster oral intake during feeding. Thus, the data indicate that desaturation after feeding remains a recurrent problem for survivors of BPD after discharge. Individual approaches that incorporate parental education and behavioral interventions might decrease the risk of significant hypoxemia during oral feeding in infants with BPD.

Intrinsic PEEP Monitored in the Ventilated ARDS Patient with a Mathematical Method—L Eberhard, J Guttmann, G Wolff, W Bertschmann, A Minzer, HJ Kohl, et al. J Appl Physiol 1992;73(2):479.

Under mechanical volume-controlled ventilation, the intensive care patient can develop intrinsic positive end-expiratory pressure (iPEEP); that is, the passive expiration is terminated by the following inspiration before the alveolar pressure comes to its physical equilibrium value. We present a mathematical method to estimate this alveolar dynamic iPEEP breath by breath, without the need of a maneuver. We tested it in paralyzed patients ventilated for adult respiratory distress syndrome after multiple trauma and/or sepsis, and we compared the results obtained with the new mathematical method with those from the occlusion method introduced by Pepe and Marini. The results agreed well (median difference of 0.8 mbar in 201 investigations in 12 patients). However, the mathematically determined values, representing dynamic iPEEP, are systematically slightly smaller than those measured by the occlusion maneuver. A variation of expiratory time suggests that this difference might be due to mechanical time-constant inhomogeneity, viscoelastic processes, or other mechanisms showing time dependence.

Functional Residual Capacity and Ventilation Homogeneity in Mechanically Ventilated Small Neonates—CT Vilstrup, LJ Bjorklund, A Larsson, B Lachmann, O Werner. J Appl Physiol 1992;73(1):276.

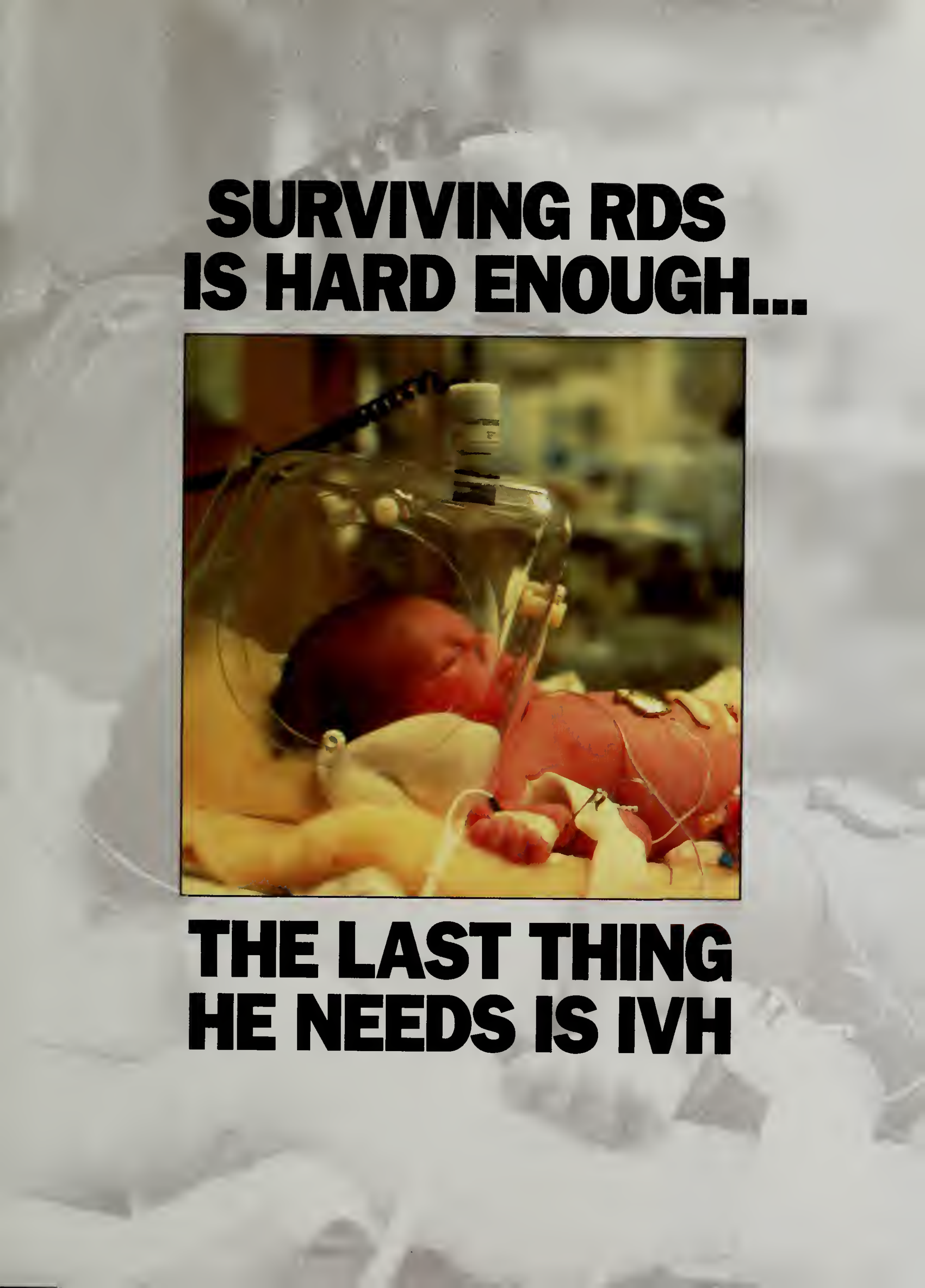
A modification of a computerized tracer gas (SF_6) washout method was designed for serial measurements of functional residual capacity (FRC) and ventilation homogeneity in mechanically ventilated very-low birth-weight infants with tidal volumes down to 4 mL. The method, which can be used regardless of the inspired O_2 concentration, gave accurate and reproducible results in a lung model and good agreement compared with He dilution in rabbits. FRC was measured during 2-4 cm H_2O of positive end-expiratory pressure (PEEP) in 15 neonates (700-1,950 g), most of them with mild-to-moderate respiratory distress syndrome. FRC increased with body weight and decreased ($p < 0.05$) with increasing O_2 requirement. Change to zero end-expiratory pressure caused an immediate decrease in FRC by 29% ($p < 0.01$) and gave FRC (mL) = $-1.4 + 17 \times$ weight (kg) ($r = 0.83$). Five minutes after PEEP was discontinued ($n = 12$), FRC had decreased by a further 16% ($p < 0.01$). The washout curves indicated a near-normal ventilation homogeneity not related to changes in PEEP. This was interpreted as evidence against the presence of large volumes of trapped alveolar gas.

Oro-Naso-Pharyngeal Suction at Birth: Effects on Respiratory Adaptation of Normal Term Vaginally Born Infants—PC Estol, H Piriz, S Basalo, F Simini, C Grela. J Perinat Med 1992;20:297.

The effect of oro-naso-pharyngeal suction at birth on pulmonary mechanics is described in a random assigned controlled study of 40 normal term vaginally born infants. Twenty cases had their oro-naso-pharynx suctioned immediately after birth (S Group), whereas 20 were not suctioned in the neonatal period (NS Group). A computerized pneumotachographic system (MECVENT) was used for the assessment of respiratory mechanics (dynamic compliance, C_{dyn}) and total pulmonary resistance (R) in inspiration and expiration at 10, 30, and 120 minutes after birth. In both groups the C_{dyn} increased during the study period whereas the R decreased, mainly in the initial 30 minutes. No significant differences were observed between S and NS groups for any of the parameters of respiratory mechanics. The results obtained in this study provide no physiological basis to recommend routine airway suction at birth in normal, term, vaginally born infants.

High-Dose Epinephrine in Adult Cardiac Arrest—IG Stiell, PC Hebert, BN Weitzman, GA Wells, S Raman, RM Stark, et al. N Engl J Med 1992;327:1045.

BACKGROUND: Recent studies suggest that doses of epinephrine of 0.1 mg per kilogram of body weight or higher may improve myocardial and cerebral blood flow as well as survival in cardiac arrest. Such studies have called into question the traditional dose of epinephrine (0.007 to 0.014 mg per kilogram) recommended for advanced cardiac life support. METHODS: We randomly assigned 650 patients who had had cardiac arrest either in or outside the hospital to receive up to five doses of high-dose (7 mg) or standard dose (1 mg) epinephrine at five-minute intervals according to standard protocols for advanced cardiac life support.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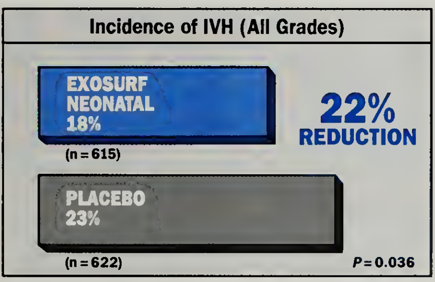
REDUCE THE RISK OF CEREBRAL HEMORRHAGE

Significant IVH Reduction in Infants ≥1250 g

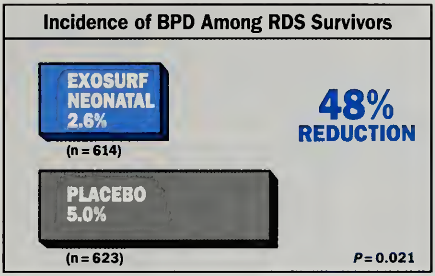
Despite successes in improving the survival of infants with RDS, intraventricular hemorrhage (IVH) remains a serious problem.[3-5] However, in a recently reported placebo-controlled rescue trial in infants ≥1250 g, EXOSURF Neonatal actually *reduced* the incidence of IVH.[2] In trials of smaller infants (<1250 g), EXOSURF Neonatal has never been observed to significantly increase IVH.



Two-dose treatment. Incidence of IVH in infants weighing 1250 g or more.
Adapted from Long et al[2 (p1700)]

Significant BPD Reduction in Infants ≥1250 g

In the rescue trial involving over 1200 infants, administration of EXOSURF Neonatal significantly reduced the incidence of bronchopulmonary dysplasia (BPD) by nearly 50% (*P*=0.021).



Two-dose treatment. Incidence of BPD in 28-day survivors following RDS in infants weighing 1250 g or more.
Adapted from Long et al[2 (p1699)]

No Increase in Sepsis

No difference in the incidence of sepsis has been seen with EXOSURF Neonatal during placebo-controlled trials (n=1517). The rate of sepsis was similar in an open trial of 11,455 infants.[6]

No Animal Proteins

EXOSURF is purely synthetic and carries no known infectious or immunologic risks.

Other Safety Considerations

Various forms of pulmonary air leak were reduced in all controlled trials. A single controlled study in infants 500-699 g reported a significant increase in pulmonary hemorrhage.[7] Significant increases in apnea were reported in three controlled trials.[1,2,8] Apnea appears to be a marker for improved survival.[2]

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*Increased pulmonary hemorrhage was noted in one trial of infants 500-699 g[7]; increased apnea has been noted in some trials.[1,2,8]

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PLEASE CONSULT FULL PRODUCT INFORMATION BEFORE PRESCRIBING

INDICATIONS AND USAGE: Exosurf Neonatal is indicated for: 1. **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS), 2. **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity, and 3. **Rescue** treatment of infants who have developed RDS.

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf Neonatal.

WARNINGS: Intratracheal Administration Only: Exosurf Neonatal should be administered only by instillation into the trachea (see DOSAGE AND ADMINISTRATION). **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal. **Acute Effects:** Exosurf Neonatal can rapidly affect oxygenation and lung compliance. **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak. **Hyperoxia:** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment. Failure to reduce FiO_2 in such instances can result in hyperoxia. **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow. **Pulmonary Hemorrhage:** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, fatal pulmonary hemorrhage occurred in three infants; two in the Exosurf Neonatal group and one in the placebo group. Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. **Mucous Plugs:** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately.

PRECAUTIONS: General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry. However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered. The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection. **Prophylactic Treatment—Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly: improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS). Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups. Data on multiple doses in infants in this weight class are not yet available. **Rescue Treatment—Number of Doses:** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 µg/plate was not mutagenic in the Ames Salmonella assay. Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal. The effects of Exosurf Neonatal on fertility have not been studied.

ADVERSE REACTIONS:
General: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died. In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made. In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased. A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. ***Reflux:*** Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes.

DOSAGE AND ADMINISTRATION: Preparation of Suspension: Exosurf Neonatal is best reconstituted immediately before use because it does not contain antibacterial preservatives. However, the reconstituted suspension is chemically and physically stable when stored at 2° to 30°C (36° to 86°F) for up to 12 hours following reconstitution. Solutions containing buffers or preservatives should not be used for reconstitution. **Do Not Use Bacteriostatic Water for Injection, USP.** Each vial of Exosurf Neonatal should be reconstituted only with **8 mL** of the accompanying diluent (preservative-free Sterile Water for Injection). **Dosage: Accurate determination of weight at birth is the key to accurate dosing.** ***Prophylactic Treatment:*** The first dose of Exosurf Neonatal should be administered as a single 5 mL/kg dose as soon as possible after birth. Second and third doses should be administered approximately 12 and 24 hours later to all infants who remain on mechanical ventilation at those times. ***Rescue Treatment:*** Exosurf Neonatal should be administered in two 5 mL/kg doses. The initial dose should be administered as soon as possible after the diagnosis of RDS is confirmed. The second dose should be administered approximately 12 hours following the first dose, provided the infant remains on mechanical ventilation. **Use of Special Endotracheal Tube Adapter:** With each vial of Exosurf Neonatal for Intratracheal Suspension, five different sized endotracheal tube adapters each with a special right angle Luer®-lock sideport are supplied. The adapters are clean but not sterile. **Administration:** The infant should be suctioned prior to administration of Exosurf Neonatal. Exosurf Neonatal suspension is administered via the sideport on the special endotracheal tube adapter **WITHOUT INTERRUPTING MECHANICAL VENTILATION.** Each Exosurf Neonatal dose is administered in two 2.5 mL/kg half-doses. Each half-dose is instilled slowly over 1 to 2 minutes (30 to 50 mechanical breaths) in small bursts timed with inspiration. After the first 2.5 mL/kg half-dose is administered in the midline position, the infant's head and torso are turned 45° to the **right** for 30 seconds while mechanical ventilation is continued. After the infant is returned to the midline position, the second 2.5 mL/kg half-dose is given in an identical fashion over another 1 to 2 minutes. The infant's head and torso are then turned 45° to the **left** for 30 seconds while mechanical ventilation is continued, and the infant is then turned back to the midline position. These maneuvers allow gravity to assist in the distribution of Exosurf Neonatal in the lungs. During dosing, heart rate, color, chest expansion, facial expressions, the oximeter, and the endotracheal tube patency and position should be monitored. **Suctioning should not be performed for two hours after Exosurf Neonatal is administered, except when dictated by clinical necessity.**

HOW SUPPLIED: Exosurf Neonatal for Intratracheal Suspension is supplied in a carton containing one 10 mL vial of Exosurf Neonatal for Intratracheal Suspension, one 10 mL vial of Sterile Water for Injection, and five endotracheal tube adapters (2.5, 3.0, 3.5, 4.0, and 4.5 mm I.D.). (NDC 0081-0207-01). Store Exosurf Neonatal for Intratracheal Suspension at 15° to 30°C (59° to 86°F) in a dry place.

EDUCATIONAL MATERIAL: A videotape on dosing is available from your Burroughs Wellcome Co. representative. This videotape demonstrates techniques for safe administration of Exosurf Neonatal and should be viewed by health care professionals who will administer the drug.

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References: 1. Long W, Thompson T, Sundell H, et al. Effects of two rescue doses of a synthetic surfactant on mortality rate and survival without bronchopulmonary dysplasia in 700- to 1350-gram infants with respiratory distress syndrome. *J Pediatr.* 1991;118:595-605. **2.** Long W, Corbet A, Cotton R, et al. A controlled trial of synthetic surfactant in infants weighing 1250 g or more with respiratory distress syndrome. *N Engl J Med.* 1991;325:1696-1703. **3.** Speer CP, Robertson B, Curstedt T, et al. Randomized European multicenter trial of surfactant replacement therapy for severe neonatal respiratory distress syndrome: single versus multiple doses of Curosurf. *Pediatrics.* 1992;89:13-20. **4.** Horbar JD, Soll RF, Schachinger H, et al. A European multicenter randomized controlled trial of single dose surfactant therapy for idiopathic respiratory distress syndrome. *Eur J Pediatr.* 1990;149:416-423. **5.** Cowan F, Whitelaw A, Wertheim D, Silverman M. Cerebral blood flow velocity changes after rapid administration of surfactant. *Arch Dis Child.* 1991;66:1105-1109. **6.** Russell L, White A, Andrews E, et al. Observational study of synthetic surfactant in 11,455 infants. Presented at the 1992 Meeting of the American Pediatric Society/Society for Pediatric Research; May 4-7, 1992; Baltimore, MD. **7.** Stevenson D, Walther F, Long W, et al. Controlled trial of a single dose of synthetic surfactant at birth in premature infants weighing 500 to 699 grams. *J Pediatr.* 1992;120:S3-S12. **8.** Corbet A, Bucciarelli R, Goldman S, et al. Decreased mortality rate among small premature infants treated at birth with a single dose of synthetic surfactant: a multicenter controlled trial. *J Pediatr.* 1991;118:277-284

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Patients who collapsed outside the hospital received no advanced-life-support measures other than defibrillation before reaching the hospital. RESULTS: There was no significant difference between the high-dose group (n = 317) and the standard-dose group (n = 333) in the proportions of patients who survived for one hour (18 percent vs 23 percent, respectively) or who survived until hospital discharge (3 percent vs 5 percent). Among the survivors, there was no significant difference in the proportions who remained in the best category of cerebral performance (90% vs 94%) and no significant difference in the median MiniMental State score (36 vs 37). The exploration of clinically important subgroups, including those with out-of-hospital arrest (n = 335) and those with in-hospital arrest (n = 315), failed to identify any patients who appeared to benefit from high-dose epinephrine and suggested that some patients may have worse outcomes after high-dose epinephrine. CONCLUSIONS: High-dose epinephrine was not found to improve survival or neurologic outcomes in adult victims of cardiac arrest.

A Comparison of Standard-Dose and High-Dose Epinephrine in Cardiac Arrest Outside the Hospital—CG Brown, DR Martin, PE Pepe, H Stueven, RO Cummins, E Gonzalez, M Jastremski, et al, and the Multi-Center High-Dose Epinephrine Study Group. N Engl J Med 1992;327:1051.

BACKGROUND: Experimental and uncontrolled clinical evidence suggests that intravenous epinephrine in doses higher than currently recommended may improve outcome after cardiac arrest. We conducted a prospective, multicenter study comparing standard-dose epinephrine with high-dose epinephrine in the management of cardiac arrest outside the hospital. METHODS: Adult patients were enrolled in the study if they remained in ventricular fibrillation, or if they had asystole or electromechanical dissociation, at the time the first drug was to be administered to treat the cardiac arrest. Patients were randomly assigned to receive either 0.02 mg of epinephrine per kilogram of body weight (standard-dose group, 632 patients) or 0.2 mg per kilogram (high-dose group, 648 patients), both given intravenously. RESULTS: In the standard-dose group 190 patients (30%) had a return of spontaneous

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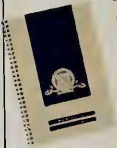

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circulation, as compared with 217 patients (33%) in the high-dose group; 136 patients (22%) in the standard-dose group and 145 patients (22%) in the high-dose group survived to be admitted to the hospital. Twenty-six patients (4%) in the standard-dose group and 31 (5%) in the high-dose group survived to discharge from the hospital. Ninety-two percent of the patients discharged in the standard-dose group and 94 percent in the high-dose group were conscious at the time of hospital discharge. None of the differences in outcome between the groups were statistically significant. CONCLUSIONS: In this study, we were unable to demonstrate any difference in the overall rate of return of spontaneous circulation, survival to hospital admission, survival to hospital discharge, or neurologic outcome between patients treated with a standard dose of epinephrine and those treated with a high dose.

Improved Outcome of Respiratory Syncytial Virus Infection in a High-risk Hospitalized Population of Canadian Children—L Navas, E Wang, V de Carvalho, J Robinson, and the Pediatric Investigators Collaborative Network on Infections in Canada. J Pediatr 1992;121(3):348.

PURPOSE: To determine the outcomes in children at high risk for death or complications from respiratory disease who are hospitalized with respiratory syncytial virus (RSV) infection. DESIGN: Retrospective chart review. SETTING: Twelve pediatric tertiary care centers. PATIENTS: All hospitalized children with an RSV infection diagnosed by a positive antigen detection test result or viral isolation during the study period from 1988 to 1991, encompassing three winter seasons. Charts from patients in the following high-risk groups were reviewed in detail: (1) congenital heart disease, (2) chronic lung disease, (3) immunodeficiency, (4) age < 6 weeks, (5) gestational age < 36 weeks, and (6) hypoxia defined as oxygen saturation < 90% or arterial oxygen pressure < 60 mm Hg). MEASUREMENTS. The age of all children, the date of RSV identification, and the use of oxygen supplementation, intensive care, and ventilatory support. In addition, the duration of these treatments and the duration of hospitalization were noted. Left-to-right shunting and pulmonary hypertension before RSV infection were determined in those children with congenital heart disease. The nature of the chronic lung disease was noted. Death within 2 weeks of RSV identification was recorded, and the use of ribavirin, bronchodilators, and corticosteroids was determined. RESULTS: Significant year-to-year variation in the frequency of RSV infection was confirmed, with a peak during the 1989-1990 winter noted by the majority of centers ($p = 0.0001$). Of the 1,584 patients in the study, 260 had underlying cardiac disease, 200 had chronic lung disease, 35 had compromised immune function, 378 had been premature, 373 were < 6 weeks to age, and 338 had hypoxia. Seventeen patients died within 2 weeks (mortality rate 1%); significantly more patients with underlying cardiac disease (3.4%) or lung disease (3.5%) died. Immunocompromised patients had the longest hospital stay (median 39 days), followed by those patients with underlying cardiac or pulmonary disease.

Respiratory Syncytial Virus Morbidity and Mortality Estimates in Congenital Heart Disease Patients: A Recent Experience—FW Moler, AS Khan, JN Meliones, JR Custer, J Palmisano, TC Shope. Crit Care Med 1992;20:1406. *Related Editorial: Pollack MM. An RSVP to RSV (Respiratory Syncytial Virus): Declining Mortality Rates. Crit Care Med 1992;20(10):1375.*

OBJECTIVE: To determine recent morbidity and mortality rates from respiratory syncytial virus infection in a pediatric congenital heart disease population. DESIGN: Retrospective cohort study design. SETTING: The CS Mott Children's Hospital University of Michigan Medical Center. PATIENTS: A total of 740 pediatric patients hospitalized at the University of Michigan Medical Center for symptomatic respiratory syncytial virus infection, of whom, 79 patients had clinically important congenital heart disease. INTERVENTIONS: None. MEASUREMENTS & MAIN RESULTS: We retrospectively examined the charts of 740 patients hospitalized at our children's hospital from July 1, 1983 to June 30, 1990 with symptomatic respiratory syncytial virus infection to assess morbidity and mortality outcomes. Seventy-nine patients had congenital heart disease and 40 of these patients had pulmonary hypertension. For the entire cohort and a subset of patients with community-acquired infection, those patients with congenital heart disease had longer durations of hospitalization and greater need for, and days of, both intensive care and mechanical ventilation than patients without congenital heart disease. Mortality risk for respiratory syncytial virus community-acquired infection was not different for congenital heart disease vs non-congenital heart disease patients (0.0% vs 0.2%; $p = 1.00$). When examining only patients with congenital heart disease, those patients with pulmonary hypertension had increased hospital days and greater intensive care and mechanical ventilation durations compared with patients without this diagnosis. The overall mortality rate was low and was equally low for congenital heart disease groups with or without pulmonary hypertension (2.6 vs 2.6). For community-acquired illness, no mortality was found in either congenital heart disease group.

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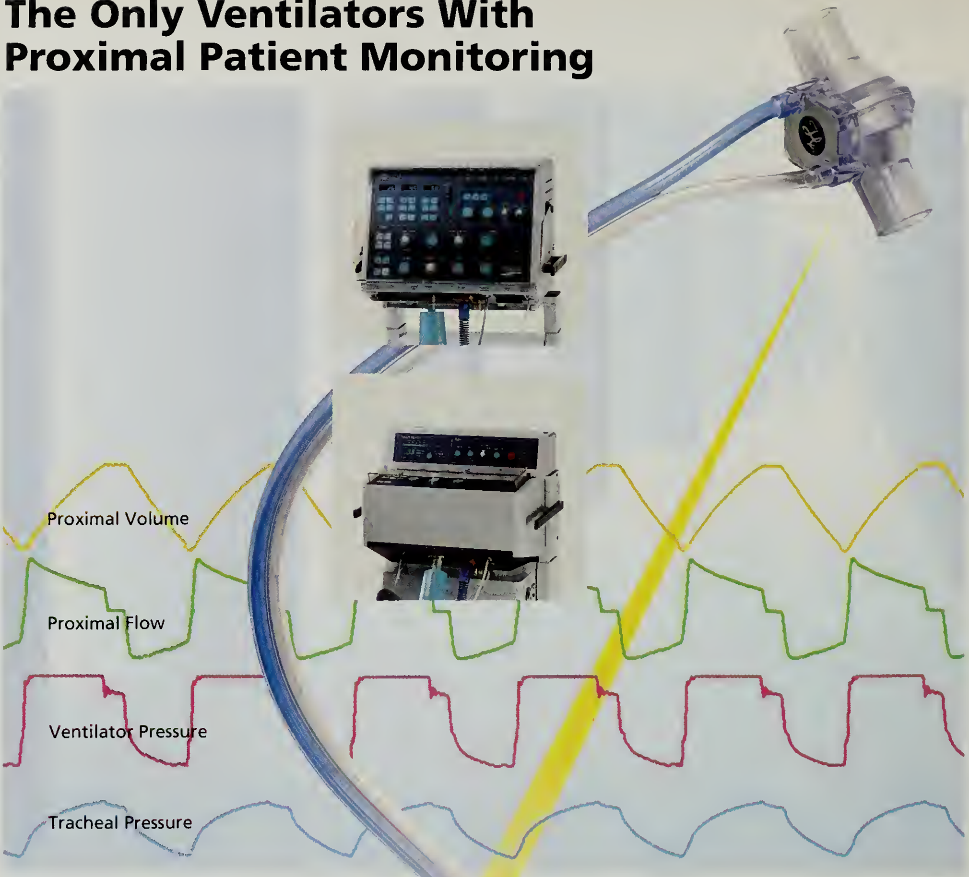

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When the cohort of congenital heart disease patients was divided into pre- and postribavirin administration eras, no differences in mean hospital duration, ICU days, and mechanical ventilation days were noted. Of the 79 congenital heart disease patients, only two died during their hospitalization in which respiratory syncytial virus infection occurred. Both patients had nosocomial-acquired respiratory syncytial virus and both were from the postribavirin administration cohort. One of these two patients had received antiviral therapy. Neither death was secondary to respiratory syncytial virus respiratory failure (based on pathologic examination). CONCLUSIONS: We conclude that respiratory syncytial virus mortality risk in pediatric patients with congenital heart disease is less than the risk reported a decade ago. Respiratory syncytial virus infection in congenital heart disease patients with pulmonary hypertension is associated with increased morbidity but not increased mortality rates. The markedly decreased respiratory syncytial virus mortality risk in patients with congenital heart disease currently experienced is likely secondary to improvements in intensive care management and advances in the surgical correction in this population rather than antiviral therapy.

Mean Airway Pressure: Physiologic Determinants and Clinical Importance—Part 1: Physiologic Determinants and Measurements—JJ Marini, SA Ravenscraft. Crit Care Med 1992;20:1461.

PURPOSES: To discuss the theoretical relationship of mean alveolar pressure to its most easily measured analog, the mean airway pressure, and to describe the key determinants, measurement considerations, and clinical implications of this index. DATA SOURCES: Relevant articles from the medical and physiologic literature, as well as mathematical arguments developed in this article from first principles. STUDY SELECTION: Theoretical, experimental, and clinical information that elucidates the physiologic importance, measurement, or adverse consequences of mean airway pressure. DATA EXTRACTION: Mathematical models were used in conjunction with data from the published literature to develop a unified description of the physiological and clinical relevance of mean airway pressure. SYNTHESIS: Geometrical and mathematical analyses demonstrate that shared elements comprise mean airway pressure and mean alveolar pressure, two variables that are related by the formula: mean alveolar pressure = mean airway pressure + $(\dot{V}_E/60) \times (R_E - R_I)$, where $\dot{V}_E$, R_E, and R_I are minute ventilation and expiratory and inspiratory resistances, respectively. Clear guidelines can be developed for selecting the site of mean airway pressure determination, for specifying technical requirements for mean airway pressure measurement, and for delineating clinical options to adjust the level of mean airway pressure. Problems in viewing mean airway pressure as a reflection of mean alveolar pressure can be interpreted against the theoretical basis of their interrelationship. In certain settings, mean airway pressure closely relates to levels of ventilation, arterial oxygenation, cardiovascular function, and barotrauma. Because mean airway pressure is associated with both beneficial and adverse effects, a thorough understanding of its theoretical and practical basis is integral to formulating an effective pressure targeted strategy of ventilatory support. CONCLUSIONS: Mean airway pressure closely reflects mean alveolar pressure, except when flow-resistive pressure losses differ greatly for the inspiratory and expiratory phases of the ventilatory cycle. Under conditions of passive inflation, mean airway pressure correlates with alveolar ventilation, arterial oxygenation, hemodynamic performance, and barotrauma. We encourage wider use of this index, appropriately measured and interpreted, as well as its incorporation into rational strategies for the ventilatory management of critical illness.

A Controlled Trial of Scheduled Replacement of Central Venous and Pulmonary-Artery Catheters—DK Cobb, KP High, RG Sawyer, CA Sable, RB Adams, DA Lindley, et al. N Engl J Med 1992;327:1062.

BACKGROUND: The incidence of infection increases with the prolonged use of central vascular catheters, but it is unclear whether changing catheters every three days, as some recommend, will reduce the rate of infection. It is also unclear whether it is safer to change a catheter over a guide wire or insert it at a new site. METHODS: We conducted a controlled trial in adult patients in intensive care units who required central venous or pulmonary-artery catheters for more than 3 days. Patients were assigned randomly to undergo one of four methods of catheter exchange: replacement every three days either by insertion at a new site (Group 1) or by exchange over a guide wire (Group 2), or replacement when clinically indicated either by insertion at a new site (Group 3) or by exchange over a guide wire (Group 4). RESULTS: Of the 160 patients, 5% had catheter-related bloodstream infections, 16% had catheters that became colonized, and 9% had major mechanical complications. The incidence rates (per 1,000 days of catheter use) of bloodstream infection were 3 in Group 1, 6 in Group 2, 2 in Group 3, and 3 in Group 4; the incidence rates of

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mechanical complications were 14, 4, 8, and 3, respectively. Patients randomly assigned to guide-wire assisted exchange were more likely to have bloodstream infection after the first three days of catheterization (6% vs 0, $p = 0.06$). Insertions at new sites were associated with more mechanical complications (5% vs 1%, $p = 0.005$). CONCLUSIONS: Routine replacement of central vascular catheters every 3 days does not prevent infection. Exchanging catheters with the use of a guide wire increases the risk of bloodstream infection, but replacement involving insertion of catheters at new sites increases the risk of mechanical complications.

Infectious and Mechanical Complications of Central Venous Catheters Placed by Percutaneous Venipuncture and over Guidewires—MT Hagley, B Martin, P Gast, SM Traeger. Crit Care Med 1992;20:1426.

OBJECTIVE: To compare the frequency of infectious and mechanical complications of central venous and pulmonary artery catheters placed by initial venipuncture vs over a guidewire at existing sites. HYPOTHESIS: Exchange of central venous catheters and pulmonary artery catheters over a guidewire as opposed to fresh venipuncture reduces mechanical complications without increasing risk of infection. DESIGN: Chart audit. PATIENTS: Medical, surgical, and coronary ICU patients requiring invasive monitoring or central venous access. INTERVENTIONS: Patients requiring prolonged catheterization underwent periodic exchange of catheters over a guidewire. Rates of catheter related infections and mechanical complications were determined for central venous catheters placed by initial venipuncture and those catheters placed by guidewire exchange. MEASUREMENTS & MAIN RESULTS: Over a 12-mo period, 939 catheters were inserted in 454 patients. Of these 939 catheters, 534 were placed by guidewire exchange. Use of a guidewire was associated with a decreased frequency of pneumothorax and hemothorax compared with initial venipuncture (0/405 [0%] vs 7/534 [1.3%], respectively; $p < 0.05$) but not with increased risk of infection (9/405 [2.2%] vs 14/534 [2.6%], respectively; NS). Guidewire-facilitated replacement of multiple consecutive catheters at the same site did not increase the risk of catheter-

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related infection. Catheters placed via internal jugular veins were more likely to become infected than catheters placed via subclavian veins (17/477 [3.6%] vs 3/430 [0.7%], respectively; $p < 0.01$). CONCLUSIONS: When prolonged central venous or pulmonary artery catheterization is necessary, periodic catheter replacement over a guidewire is associated with fewer mechanical complication than initial venipuncture. Periodic catheter replacement over a guidewire is also associated with no increase in risk of infection.

Inhaled Nitric Oxide in Persistent Pulmonary Hypertension of the Newborn—JD Roberts, DM Polaner, P Lang, WM Zapol. Lancet 1992;340:818.

Nitric oxide (NO) has vasodilatory effects on the pulmonary vasculature in adults and animals. We examined the effects on systemic oxygenation and blood pressure of inhaling up to 80 parts per million by volume of NO at F_{IO_2} 0.9 for up to 30 minutes by 6 infants with persistent pulmonary hypertension of the newborn (PPHN). In all infants this treatment rapidly and significantly increased preductal oxygen saturation (S_{pO_2}); in 5 infants postductal S_{pO_2} and oxygen tensions also increased. Inhalation of NO did not cause systemic hypotension or raise methaemoglobin. These data suggest that low levels of inhaled NO have an important role in the reversal of hypoxaemia due to PPHN.

Normobaric Measurement of Arterial Oxygen Tension in Subjects Exposed to Hyperbaric Oxygen—LK Weaver, S Howe. Chest 1992; 102:1175.

This study demonstrates the ability of an automated blood gas analyzer (Radiometer ABL 330) operated at atmospheric pressure to measure the arterial oxygen tension (P_{aO_2}) of ten healthy volunteers exposed to hyperbaric oxygen (HBO_2) up to 3.0 atmospheres absolute. Arterial blood samples were aspirated from subjects compressed in a single-person hyperbaric chamber amd were analyzed immediately in the blood gas analyzer. The subjects' values for P_{aO_2} correlated with the calculated alveolar O_2 tension (P_{AO_2}) (measured $P_{aO_2} = 0.827 \times P_{AO_2} - 15.1$) ($r^2 = 0.97$). Tonometric experiments indicated a difference between saline and blood P_{O_2} measurements. We therefore derived a correction factor for blood measurements (corrected $P_{aO_2} = 0.908 \times P_{AO_2} - 52.4$) ($r^2 = 0.98$). These results compared favorably with P_{aO_2} measurements made with blood gas analyzers calibrated inside walk-in hyperbaric chambers. We conclude that the P_{aO_2} of normal subjects exposed to HBO_2 can be measured accurately at atmospheric pressure with this automated blood gas analyzer. Prior to this study, hyperbaric P_{aO_2} measurements could only be performed within walk-in chambers. Our observations generalize the normobaric measurement of hyperbaric P_{aO_2} to patients treated in single-person and walk-in chambers.

Placebo-Controlled Trial of Prednisolone in Children Intubated for Croup—J Tibballs, FA Shann, LI Landau. Lancet 1992;340:745.

Many studies have attempted to find out whether steroid treatment is beneficial in children with croup, but the results have been inconclusive. We have done a prospective placebo-controlled study of the effect of prednisolone on two clinical endpoints—the duration of intubation and the need for reintubation. Reasons for exclusion were age under 6 months, congenital airway anomalies, and previous intubation. 70 eligible children were randomly assigned treatment with prednisolone 1 mg/kg ($n = 38$) or placebo ($n = 32$) every 12 h given by nasogastric tube until 24 h after extubation. 11 (34%) placebo-treated and only 2 (5%) prednisolone-treated patients required reintubation after accidental or elective extubation ($p = 0.004$, Fisher's exact test; odds ratio 8.9, 95% confidence interval 1.7-59.3). Survival analysis with log-normal regression showed that the duration of intubation was shorter with steroid therapy ($p < 0.003$) and increasing age ($p < 0.02$), but was not influenced by endotracheal tube size or abnormality on chest radiograph. The median duration of intubation was 138 (95% CI 118-160) h in children who received placebo and 98 (85-113) h in the prednisolone group. Steroid therapy reduces the duration of intubation and the need for reintubation in children intubated for croup.

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Retrospectroscope Redux: Peering Once More into Dr Comroe's Wonderful Instrument

In the mid-1960s, an inhalation therapy department's library, if it existed, probably offered four to six reference texts. Of these, the 'big four' often were referred to by the colors of their covers:

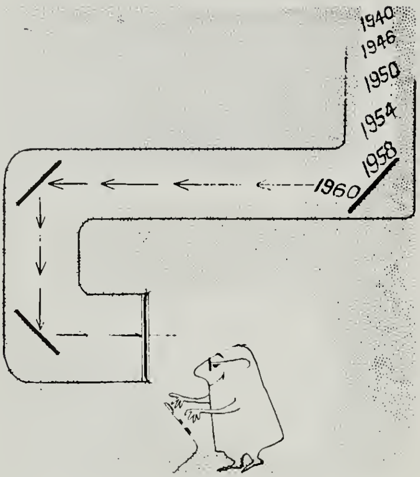
> "Hey, Joe. Is it residual *capacity*—or residual *volume?*
>
> "I forget. Look in the brown book—or the green one."

If you were feeling semi-formal, then you'd call the book by the name of its principal author. The blue one was "Safar."[1] The brown cover was "Comroe."[2] Green was another "Comroe,"[3]—and red was "Bendixen."[4] As can be seen, half of the 'big four' were books by Comroe. They were *The Lung* [its short title], by Comroe and others, and *Physiology of Respiration,* by Comroe alone. The latter, especially, was (and is) a model textbook: good organization, clear writing, useful graphics. Thus, Comroe's name came up pretty often in those days. Now, more than 25 years later, in this issue of RESPIRATORY CARE we're bringing it up again.

Julius H Comroe Jr MD (to give him the full treatment) was a pioneer and giant in pulmonary physiology whose contributions to the literature appeared from the 1930s to 1980. The descriptors "giant" and "pioneer" do not seem excessive when you look at some of the highlights of Comroe's publications. Proceeding chronologically, we find him writing on aortic chemoreceptors (1939),[5] chemical and electrical stimulation of the respiratory center (1943),[6] the hyperpnea of exercise (1944),[7] oxygen toxicity at sea level and at 18,000 feet (1945),[8] artificial respiration (1946),[9] pulmonary function tests (1950),[10] standardized definitions and symbols in respiratory physiology (1950)[11] (our colleague Fred Helmholz—still active today—was a coauthor), mental changes in chronically hypoxemic patients during oxygen therapy (1950),[12] the physiologic basis for oxygen therapy (1950),[13] detection of uneven alveolar ventilation during a single breath of oxygen (1951),[14] a single method of concurrent measurement of compliance and resistance to breathing (1954),[15] dyspnea (1956),[16] tissue oxygen tension (1957),[17] design of a body plethysmograph for studying cardiopulmonary physiology (1959),[18] the brown book (1959 and 1962),[2] tobacco and health (1962),[19] the unreliability of cyanosis in the recognition of arterial hypoxemia (1964),[20] and the green book (1965).[3]

Obviously, Dr Comroe was interested in every aspect of pulmonary physiology. In addition, he was much involved in medical education, serving as Professor of Physiology, first at the Graduate School of Medicine in Philadelphia and later at the University of California in San Francisco. At the latter medical center he also founded and long directed the world-famous Cardiovascular Research Institute, popularly known as the CVRI.

As a medical educator, in 1961 Comroe was Chairman of that year's gathering called the American Medical College's Institute. His introductory remarks to the Institute included some comments on recent changes in facilities for research. "Scientists who formerly merely observed," he said, "now use elaborate and expensive apparatus such as the fluoroscope, the electron microscope, the ultraviolet microscope, the mass spectrometer, the infrared spectroscope, and the cathode ray oscilloscope . . . though some, I am told, still depend largely on the horoscope and retrospectroscope."[21] When Comroe's introduction was published, a sketch of the retrospectroscope was included, which is reproduced here.



Dr Comroe's Retrospectroscope. Reproduced, with permission, from J Med Educ, Dec 1962, Part 2, Page 6. (The former Journal of Medical Education is now called Academic Medicine.)

After briefly being glimpsed in the early 1960s, the retrospectroscope—a wonderful device for looking into the past—disappeared from view (so far as I know) for more than a dozen years.

However, the retrospectroscope had not been forgotten. In 1975, Comroe, now retired from the CVRI, found time to make use of it. To readers of the *American Review of Respiratory Disease,* he heralded the value of the retrospectroscope in these words: "I now use this instrument frequently—not to collect data, nor to establish priority for long-forgotten scientists who "did it first," nor to diminish the lustre of a later scientist who really rediscovered the same methods, facts, and concepts and is now credited with the original discovery. Rather, I use it to analyze the complex processes of biomedical discovery and rediscovery and the lag between the two, and the lag between rediscovery and full application to clinical medicine and surgery. What led to the original discovery? Was it applied promptly or was there a long lag between discovery and application? In either case, *why?* Santayana said "Those who cannot remember the past are condemned to repeat it." Can we learn from the Retrospectroscope how to identify promptly which discoveries are destined to be important for clinical advance and how to speed their application?"[22]

With that explanation of his employment of the retrospectroscope, Comroe went on in the same issue of *Am Rev Respir Dis* to report what the instrument had revealed about the use of carbon monoxide for measuring pulmonary diffusing capcity. The story ranges from 1881 to 1957. The big question is, Why, when the basic information had been reported by 1915, did it take until the mid-1950s for the single-breath CO test to become fully realized?[23]

You, sir, say you read that first Retrospectroscope feature back in 1975, but you've forgotten how the mystery worked out? And you, ma'am, say you were 10 years old in 1975 and couldn't even spell carbon monoxide? Well, we have a deal for both of you. That first Retrospectroscope is reprinted in this issue of RESPIRATORY CARE in a new feature called Classic Reprints (see Page 141).

Dr Comroe kept using his unique scope and reporting his results in *Am Rev Respir Dis* for a number of years. Now the American Lung Association has given us permission to bring back some of those Retrospectroscopes, and we plan to offer at least a dozen. You'll find that Julius Comroe was a good writer, a wit, and a person of good humor. Partly, he's in the tradition of those earlier writers on medical topics, Osler and Haldane. He also brings to mind our contemporary, Lewis Thomas (The Biology Watcher) and even James Burke, who uses a retrospectroscopic technique in his TV series *Connections* and *The Day the Universe Changed.* High praise, seriously given.

That means you can expect to enjoy yourself as you read the Retrospectroscopes in this and future issues.

Phil Kittredge RRT
Adjunct Editor

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Original Contributions

Pressure-Control Ventilation with a Puritan-Bennett 7200a Ventilator: Application of an Algorithm and Results in 14 Patients

William R Howard MBA RRT

BACKGROUND: Previously published reports imply that the beneficial effects of pressure-control ventilation (PCV) depend on the use of inverse inspiratory-to-expiratory-time ratios (I:E) greater than those used during conventional ventilation (CMV, volume-cycled assist-control or intermittent mandatory ventilation). RESEARCH QUESTIONS: Can PCV provide P_{aO_2} and P_{aCO_2} comparable to CMV at lower peak inflating pressure ($P_{aw\text{-}peak}$), positive end-expiratory pressure (PEEP), fractional concentration of inspired oxygen (F_{IO_2}), and minute ventilation ($\dot{V}_E$)? Is an inverse I:E necessary to obtain the comparable P_{aO_2} and P_{aCO_2}? SUBJECTS & METHOD: We ventilated 14 patients on PCV (via Puritan-Bennett 7200a) who had required $F_{IO_2} \geq 0.40$ and $P_{aw\text{-}peak} \geq 40$ cm H_2O on CMV for ≥ 24 hours and whose clinical condition had failed to improve as reflected by unchanged or deteriorating compliance, chest radiograph, P_{aO_2}, and P_{aCO_2}, and by stable or rising $P_{aw\text{-}peak}$, F_{IO_2}, $\dot{V}_E$, and PEEP. After the initial transition from CMV to PCV at comparable settings, we increased inspiratory time (t_i) in 0.1-s increments until V_T decreased, at which point t_i was decreased by 0.1 s and maintained. Other changes in ventilator variables were guided by blood-gas values and results of hemodynamic and noninvasive monitoring. RESULTS: Twenty-four hours after initiating PCV, statistically significant differences were seen (level of significance ≤ 0.0042 with Bonferroni's adjustment for multiple *t* tests) in $P_{aw\text{-}peak}$ (decreased, $p = 0.002$), dynamic compliance (increased, $p = 0.004$), $\dot{V}_E$ (decreased, $p = < 0.001$), F_{IO_2} (decreased, $p = 0.002$), alveolar-arterial partial pressure difference for oxygen [$P_{(A-a)O_2}$] (decreased, $p = 0.002$). The increase in P_{aO_2} ($p = 0.046$) and P_{aO_2}/F_{IO_2} ($p = 0.006$) and the decrease in ventilatory rate ($p = 0.005$) failed to reach significance. Eight patients were ventilated at $t_i \leq 50\%$, 2 patients at 51-60%, and 4 patients at 70-78%. CONCLUSIONS: (1) PCV can adequately ventilate and oxygenate patients in severe respiratory failure and (2) inversing I:E is not universally necessary during PCV. (Respir Care 1993;38:32-40.)

Introduction

Pressure-control ventilation (PCV) allows the clinician to preset the peak airway pressure ($P_{aw\text{-}peak}$) and the inspiratory time (t_i) for each mechanical breath. In this mode of ventilation, tidal volume (V_T) is variable depending upon total system compliance and airway resistance. In PCV, $P_{aw\text{-}peak}$ is reached quickly (within 40 ms with the Puritan-Bennett 7200a[1]). The ventilator continuously adjusts the flow to reach and maintain the specified pressure for the inspiratory time interval. Distribution of the inspired gas continues throughout inspiration at the prescribed $P_{aw\text{-}peak}$ and t_i. Flow delivery of the mandatory breath is described as decelerating. Under certain conditions (eg, inverse ratio ventilation), flow can reach zero prior to time cycling of the mandatory breath. If flow does not

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reach zero, inspired gas delivery continues until the preset t_i interval expires. Conversely, inspired gas delivery (ie, V_T) during volume-cycled ventilation continues throughout the inspiratory phase of the breath. Critics have charged that scientific evidence supporting the effectiveness of PCIRV is lacking, although they allow that PCV may prevent increased peak airway pressures, which may be beneficial in some patients.[2] PCV has been used for many years as a primary means of ventilating neonatal patients suffering from respiratory distress syndrome.[3] Additionally, successful application of PCV in patients in severe respiratory failure resulting from ARDS has been reported.[4-7] Those reporting on PCV in ARDS have been able to increase arterial oxygen tension (P_{aO_2}) while maintaining lower peak pressure ($P_{aw\text{-}peak}$), oxygen concentration (F_{IO_2}), minute volume ($\dot{V}_E$), and positive end-expiratory pressure (PEEP) than those used with conventional mechanical ventilation (CMV, volume-limited assist/control or intermittent mandatory ventilation). However, their reports imply that successful application of PCV depends on the use of ratios of inspiratory-to-expiratory time (I:E) markedly greater than those used with CMV (ie, not simply 'inverse,' which would be > 1:1, but rather as great as 3:1 and 4:1). Further, their protocols have called for immediate application of inverse I:E upon the change from CMV to PCV.[4-7]

In contrast, time-cycled, pressure-limited ventilation is applied to neonates at conventional I:E,[8] which led us to seek to determine whether some patients in severe respiratory failure might be effectively ventilated and oxygenated at I:E ≤ 1:1 (or at least on the order of 1.5:1 suggested by Cole et al[9] and reported by Ravizza et al in an abstract[10] at lower $P_{aw\text{-}peak}$, F_{IO_2}, $\dot{V}_E$, and PEEP than is possible with CMV. We report on our experience with PCV applied according to a pre-established algorithm in 14 patients in severe respiratory failure.

Subjects and Method

Subjects and Entry Criteria

Selection criteria were similar to those used by Tharratt et al[4] and Lain et al.[5] I studied 14 patients in severe respiratory failure from the adult respiratory distress syndrome (ARDS) who were judged to be failing to respond to CMV because (1) for 24 hours or more they had been receiving potentially toxic levels of oxygen ($F_{IO_2} \geq 0.40$) and/or had been ventilated with high pressures (≥ 40 cm H_2O, or 3.92 kPa); (2) their clinical condition was worsening or had failed to improve in the previous 24 hours on CMV (actually, our patients had failed to improve over the course of several days to several weeks prior to the application of PCV) as suggested by unchanged or deteriorating dynamic compliance (C_{dyn}), chest radiographs, P_{aO_2}, P_{aCO_2}, and by stable or rising $P_{aw\text{-}peak}$, F_{IO_2}, $\dot{V}_E$, and PEEP; and (3) in the clinical judgment of the managing physicians, improvement in oxygenation and ventilation (while reducing CMV $P_{aw\text{-}peak}$ and F_{IO_2}) would not be seen within a reasonable period of time with the continuance of CMV. All candidates had been managed with what are considered in our institution to be unacceptably high levels of $P_{aw\text{-}peak}$, F_{IO_2}, and/or PEEP and were considered at high risk for development of barotrauma. The prognosis was considered to be negative in all candidates. The final decision to use PCV rested with the managing physician, and the 14 represent those who met the criteria and whose physicians agreed to the change to PCV.

During CMV, all patients had been sedated and managed with paralyzing agents and this was continued during PCV. Prior to initiation of PCV, all patients had been ventilated on Puritan-Bennett 7200a ventilators (Puritan-Bennett Corporation, Carlsbad CA) in the volume-cycled mode with a decelerating ramp waveform.

Transition from CMV to PCV

For the transition from CMV to PCV, we duplicated CMV settings for $P_{aw\text{-}peak}$, PEEP, V_T, F_{IO_2}, respiratory rate (f), inspiratory time (t_i), and I:E inspiratory time was calculated from the CMV I:E and f, and was used as the initial t_i for PCV. The initial $P_{aw\text{-}peak}$ setting for PCV was determined by subtracting PEEP from the CMV $P_{aw\text{-}peak}$. (In PCV on the 7200a, applied $P_{aw\text{-}peak}$ = set $P_{aw\text{-}peak}$ + PEEP.) Initially, the set $P_{aw\text{-}peak}$ was adjusted to maintain the same $\dot{V}_E$ as required during CMV. The patient's exhaled V_T was monitored during the transition to PCV as a guideline to detect variation from CMV V_T.

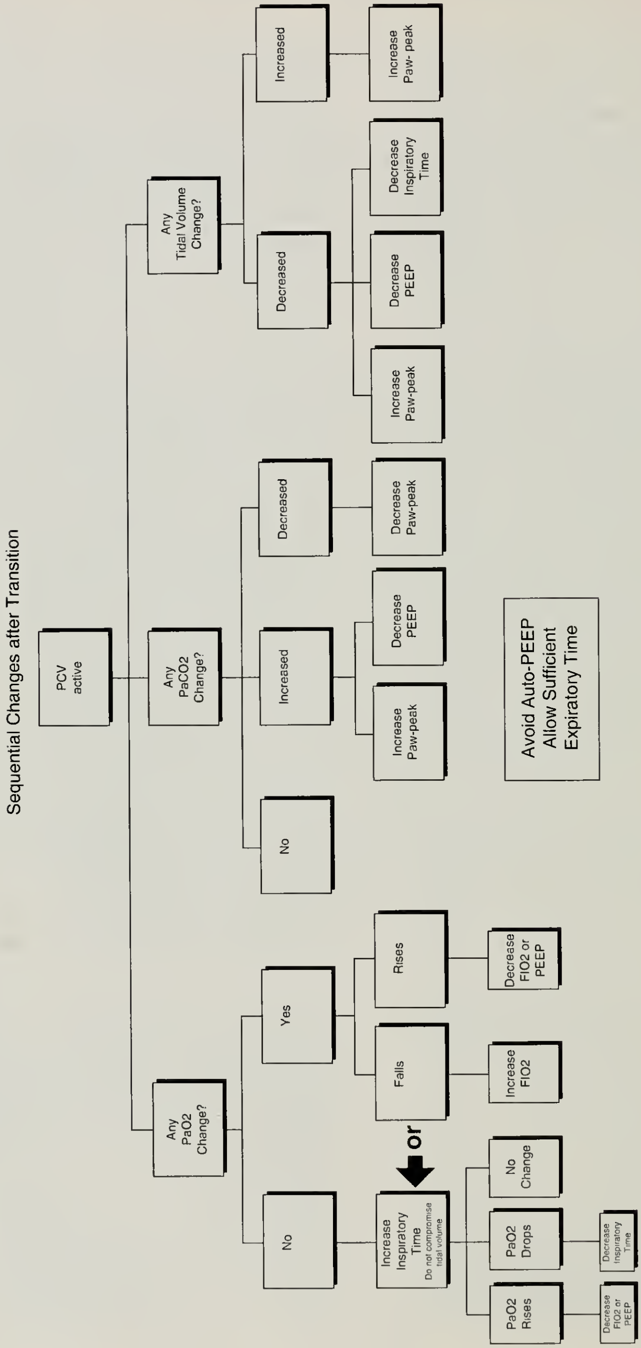

Fig. 1. Algorithm for managing patients on pressure-controlled ventilation.

Immediately after activation of PCV, $P_{aw\text{-}peak}$ was adjusted (if necessary) to ensure that exhaled V_T remained equal to CMV V_T. After this adjustment, t_i was then increased in increments of 0.1 second until exhaled V_T was observed to decrease. The t_i immediately prior to this decrease was then used as the t_i for PCV. This t_i adjustment increased $\bar{P}_{aw}$ compared to the $\bar{P}_{aw}$ during CMV. If S_{aO_2} allowed, PEEP was decreased to (1) maintain the previous $\bar{P}_{aw}$ and (2) avoid air trapping and auto-PEEP.

PCV Management

The algorithm shown in Figure 1 guided management of V_T, P_{aO_2}, and P_{aCO_2}. Ventilator settings were adjusted based on the patient's clinical data. Indexes of oxygenation [$P_{(A-a)O_2}$ or P_{aO_2}/F_{IO_2}] were used to determine changes (ie, lower F_{IO_2} with normoxia was perceived as a more desirable endpoint than 'improved' P_{aO_2}; likewise a lower P_{aCO_2} was not an endpoint, but rather a similar P_{aCO_2} at lower $P_{aw\text{-}peak}$ and V_T was considered an indication of clinical improvement.)

Intrinsic, or auto-PEEP ($PEEP_i$), was measured according to the method of Braschi[11-14] utilizing a Marquest nonrebreathing T-piece (Marquest Inc, Englewood CO, model 090-193699) on the inspiratory limb of the patient circuit.

Statistical Analysis

Differences in parameters on CMV immediately prior to transition to PCV and parameters on PCV at 24 hours would be compared and tested by Student's *t* test as performed by previous authors.[3,4,8,9] The overall significance of 12 paired sets of data was chosen at the 0.05 level. A Bonferroni adjustment[15] was made resulting in a significance level of 0.0042.

Results

The results obtained in our 14 patients are shown in Tables 1 and 2. Figure 2A-H contrasts results during CMV and PCV. During PCV, 8 patients had a $t_i\% \leq 50$, 2 patients a $t_i\%$ of 51-60, and 4 patients a $t_i\%$ of 70-78. Results after 24 hours of PCV are summarized in Table 3. We did not measure more than 2 cm H_2O [0.196 kPa] of auto-PEEP on any patient in our trial. Three patients (#5, 6, and 8) developed evidence of barotrauma during CMV that persisted into PCV. However, no new evidence of barotrauma presented during PCV.

Discussion

The use of PCV in our 14 patients, resulted in findings similar to those of other authors.[4-7,9,10] However, our results occurred with the use of lower I:E than those reported by Tharratt et al,[4] Lain et al,[5] Gurevitch et al,[6] and Cole et al,[9] who used I:E of 2:1 to 4:1. Ravizza et al[10] used I:E of 1.5:1 and were able to show a significantly lower $P_{aw\text{-}peak}$ while improving P_{aO_2}/F_{IO_2} in a PCV limited application to 6 hours.

The Servo 900C (Siemens Corp, Schaumburg IL), which these authors used, offers limited control of the I:E. The 7200a offers precise control of t_i (in increments of 0.1 s) and of I:E (in increments of 0.1 for both inspiration and expiration). The 7200a ventilators that were used in this trial were equipped with a software option that provides delivery of constant-pressure mandatory breaths in CMV and SIMV modes. During PCV mandatory breaths are delivered at a preset inspiratory pressure and t_i or I:E. Inspiratory pressure can be varied from 5-100 cm H_2O, t_i from 0.2 to 5.0 seconds, and I:E to not greater than 4:1.[1]

It should be noted that when the decelerating waveform is used with CMV, peak airway pressures differs little from plateau pressures. As we anticipated, the ventilator setting and measured values in our patients P-T (transition to PCV) did not differ significantly from the settings and values at C-T (transition from conventional ventilation) because the transition was accomplished by first matching PCV settings to CMV settings and then gradually modifying settings as described. We chose not to obtain static pressures because of possible increased risk of barotrauma and hemodynamic compromise.

Investigators[4-6,9,10] who have published their findings on pressure-based inverse ratio ventilation have stated that in ARDS patients (1) oxygenation improves and reduction in F_{IO_2} is possible and (2) the likelihood of barotrauma is reduced because of

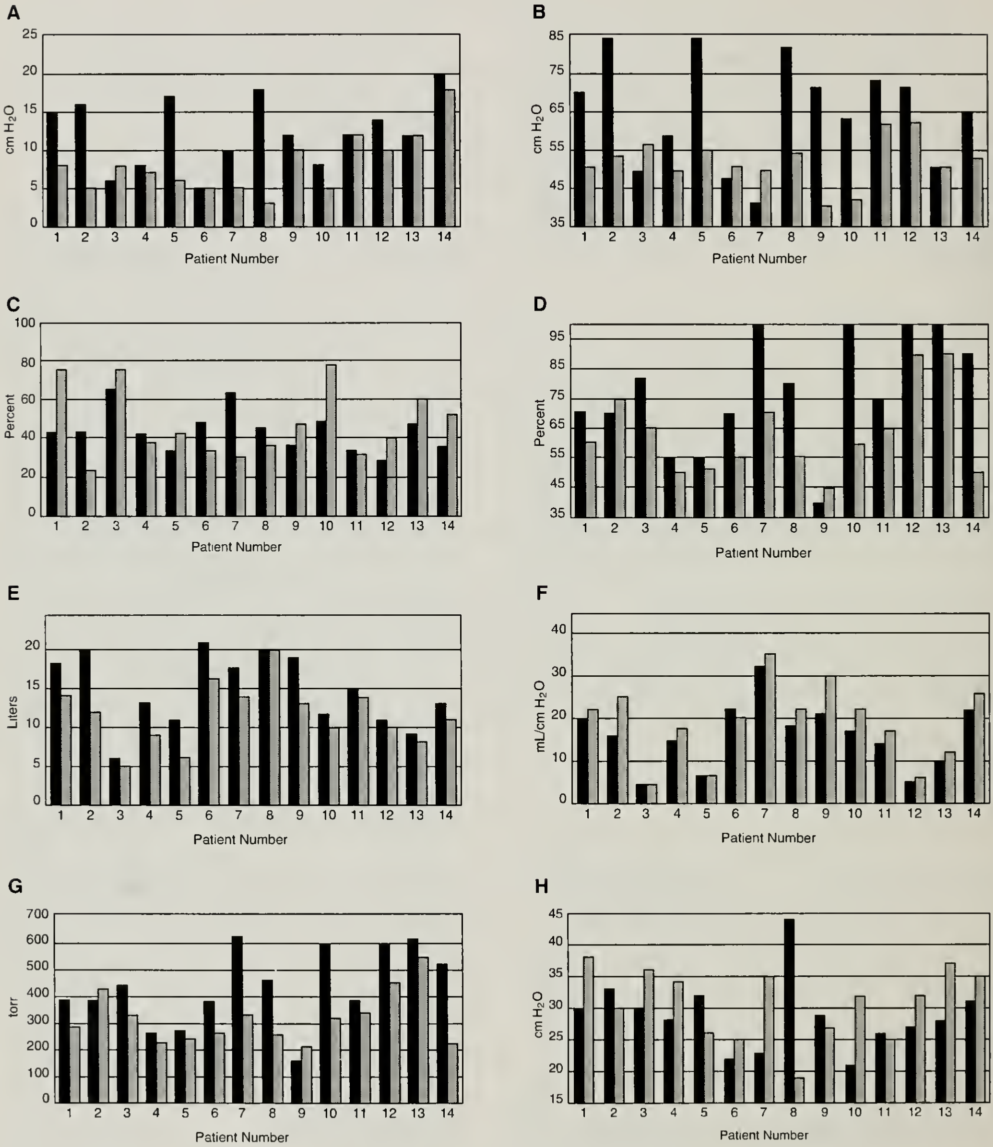

Fig. 2. Comparisons of requirements during conventional mechanical ventilation (CMV, ■) at transition and 24 hours after initiation of pressure-controlled ventilation (PCV, ▨): A. positive end-expiratory pressure, or PEEP; B. peak airway pressure, or $P_{aw\text{-}peak}$; C. percent inspiratory time, or $t_i\%$; D. fractional concentration of inspired oxygen, or F_{IO_2}; E. minute ventilation, or $\dot{V}_E$; F. dynamic compliance, or C_{dyn}; G. Alveolar-arterial oxygen partial pressure difference, or $P_{(A-a)O_2}$; and H. mean airway pressure ($\bar{P}_{aw}$).

Table 1. Ventilator Settings for 14 ARDS Patients during Conventional (CMV) and Pressure-Control Ventilation (PCV)*

Patient No.	CMV prior to PCV	Time of Measurement	V_T (mL)†	f‡	CMV PIF (L/min)	I Time (s)§	% I Time#	PEEP¶	$P_{aw\text{-}peak}$**	F_{IO_2}††	$\dot{V}_E$ (L)‡‡
1	7 w	C-24	1000	14	70	1.95	46	12	58	0.55	14
		C-T	1100	14	60	1.63	43	15	70	0.70	18
		P-T	900	16	—	2.8	75	15	58	0.70	14
		P-24	900	16	—	2.8	75	8	50	0.60	14
2	3 w	C-24	1000	20	85	1.3	43	10	60	0.55	20
		C-T	1000	20	85	1.3	43	16	82	0.70	20
		P-T	1400	20	—	1.2	40	10	77	0.70	28
		P-24	1200	10	—	1.4	23	5	53	0.75	12
3	2 w	C-24	200	30	35	1.3	65	6	45	0.85	6
		C-T	200	30	35	1.3	65	6	50	0.82	6
		P-T	290	30	—	1.3	65	6	44	0.78	9
		P-24	175	30	—	1.5	75	8	48	0.65	5
4	9 d	C-24	950	18	65	1.3	39	8	61	0.50	17
		C-T	950	14	45	1.8	42	8	58	0.55	13
		P-T	850	14	—	1.4	33	5	62	0.55	12
		P-24	700	13	—	1.7	37	7	49	0.50	9
5	3 w	C-24	420	25	30	0.8	33	15	75	0.55	10
		C-T	425	25	30	0.8	33	17	82	0.55	11
		P-T	268	26	—	1.0	43	15	50	0.53	7
		P-24	252	25	—	1.0	42	16	55	0.51	6
6	9 d	C-24	800	25	90	0.9	38	10	54	0.70	20
		C-T	950	22	90	1.3	48	5	47	0.70	21
		P-T	1000	20	—	1.3	43	5	48	0.70	20
		P-24	900	18	—	1.1	33	5	50	0.55	16
7	1 w	C-24	1000	12	85	2.3	46	10	43	0.80	12
		C-T	1000	18	85	2.1	63	10	41	1.0	18
		P-T	1150	12	—	1.5	30	15	49	1.0	14
		P-24	1200	12	—	1.5	30	15	49	0.70	14
8	1 w	C-24	1100	18	100	1.39	42	15	69	0.50	20
		C-T	1100	18	85	1.5	45	18	80	0.80	20
		P-T	1100	18	—	1.5	45	8	58	0.60	20
		P-24	1120	18	—	1.2	36	3	54	0.55	20
9	5 w	C-24	1000	14	85	1.38	32	12	69	0.40	14
		C-T	1200	16	85	1.34	36	12	70	0.40	19
		P-T	1100	14	—	1.8	42	10	51	0.40	15
		P-24	900	14	—	2.0	47	10	40	0.45	13
10	3 d	C-24	712	16	60	1.1	29	5	41	0.50	11
		C-T	880	14	75	2.1	49	8	62	1.0	12
		P-T	900	14	—	2.1	49	5	46	0.75	13
		P-24	800	12	—	3.9	78	5	41	0.60	10
11	5 d	C-24	750	18	70	1.1	33	12	62	0.8	14
		C-T	850	18	65	1.15	34	12	72	0.75	15
		P-T	750	18	—	1.0	30	12	59	0.70	14
		P-24	850	16	—	1.2	32	12	61	0.65	14
12	1 w	C-24	220	32	40	0.59	31	3	50	1.0	7
		C-T	300	36	40	0.46	28	14	70	1.0	11
		P-T	325	36	—	0.50	30	12	55	1.0	12
		P-24	300	34	—	0.7	40	10	61	0.90	10
13	3 d	C-24	400	25	45	1.1	46	13	54	1.0	10
		C-T	350	25	45	1.14	47	12	49	1.0	9
		P-T	350	25	—	1.6	67	10	50	1.0	9
		P-24	450	18	—	2.0	60	12	49	0.9	8
14	3 d	C-24	925	14	60	1.48	35	17	49	0.75	13
		C-T	950	14	60	1.48	35	20	65	0.90	13
		P-T	950	14	—	1.4	33	20	65	0.90	13
		P-24	900	12	—	2.6	52	18	52	0.50	11

*PIF = peak inspiratory flow; $\dot{V}_E$ = minute volume. C-24 = settings on CMV 24 h before transition; C-T = settings on CMV at transition; P-T = settings on PCV at transition; P-24 = settings 24 h after transition.

†Mean (SD) of V_T P-T – V_T C-T = 6 mL (149); $p = 0.89$. Mean (SD) of V_T P-24 – V_T C-T = 43 mL (152); $p = 0.3$.

‡Mean (SD) f P-T – f C-T = 0.64 (1.7); $p = 0.19$. Mean (SD) f P-24 – f C-T = 2.7 (3.0); $p = 0.005$.

§Mean (SD) t_I P-T – t_I C-T = 0.07 (0.4); $p = 0.54$. Mean (SD) t_I P-24 – t_I C-T = 0.37 (0.7); $p = 0.057$.

#Mean (SD) %t_I C-T – %t_I P-T = 0.9 (15); $p = 0.8$. Mean (SD) %t_I P-24 – %t_I C-T = 3.4 (19.2); $p = 0.29$.

¶Mean (SD) PEEP P-T – PEEP C-T = 1.75 (3.4); $p = 0.07$. Mean (SD) PEEP P-24 – PEEP C-T = 2.7 (5.2); $p = 0.07$.

**Mean (SD) $P_{aw\text{-}peak}$ C-T – $P_{aw\text{-}peak}$ P-T = 9.05 (11.3); $p = 0.01$. Mean (SD) $P_{aw\text{-}peak}$ P-24 – $P_{aw\text{-}peak}$ C-T = 13.3 (12.5); $p = 0.002$.

††Mean (SD) F_{IO_2} P-T – F_{IO_2} C-T = 4.0 (8.1); $p = 0.086$. Mean (SD) F_{IO_2} P-24 – F_{IO_2} C-T = 14.7 (14.4); $p = 0.002$.

‡‡Mean (SD) $\dot{V}_E$ P-T – $\dot{V}_E$ C-T = 0.5 (2.4); $p = 0.63$. Mean (SD) $\dot{V}_E$ P-24 – $\dot{V}_E$ C-T = 3.1 (0.24); $p < 0.001$.

Table 2. Measured Values: Comparisons between Conventional and Pressure-Control Ventilation in 14 ARDS Patients at 4 Sampling Times

Patient No. (Sex, Age)	Time of Measurement*	P_{aO_2} (torr)†	P_{aCO_2} (torr)‡	F_{IO_2}§	P_{aO_2}/F_{IO_2} (torr)#	C_{dyn} (mL/ cm H_2O¶	$P_{(A-a)O_2}$ (torr)**	$\bar{P}_{aw}$ (cm H_2O)††	Barotrauma
1 (F, 66)	C-24	62	51	0.55	113	22	266	28	No
	C-T	67	40	0.70	96	20	382	30	No
	P-T	80	43	0.70	114	21	365	44.7	No
	P-24	87	42	0.60	145	21	288	38	No
2 (M, 71)	C-24	74	44	0.55	135	20	263	26	No
	C-T	69	39	0.70	99	15	381	33	No
	P-T	68	21	0.70	97	21	405	34	No
	P-24	62	39	0.75	83	25	424	30	No
3 (F, 5)	C-24	50	75	0.85	59	5	462	32	No
	C-T	50	80	0.82	61	4	435	30	No
	P-T	85	28	0.78	109	8	436	30.2	No
	P-24	103	28	0.65	158	4	325	36	No
4 (F, 5)	C-24	58	39	0.50	116	12	250	30	No
	C-T	80	44	0.55	145	14	257	28	No
	P-T	64	34	0.55	116	14	286	39.7	No
	P-24	87	40	0.50	174	17	220	34	No
5 (M, 5)	C-24	65	40	0.55	118	7	277	28	Yes
	C-T	68	44	0.55	124	6	269	32	Yes
	P-T	82	48	0.53	155	8	236	25.8	Yes
	P-24	70	45	0.51	137	6	237	26	Yes
6 (F, 60)	C-24	68	52	0.70	97	18	366	24	Yes
	C-T	70	41	0.70	100	22	378	22	Yes
	P-T	107	38	0.70	153	23	345	22	Yes
	P-24	82	41	0.55	149	20	259	25	Yes
7 (M, 43)	C-24	59	38	0.80	74	30	464	17	No
	C-T	45	37	1.0	45	32	622	23	No
	P-T	78	37	1.0	78	34	589	22	No
	P-24	139	27	0.70	199	35	326	35	No
8 (F, 72)	C-24	72	41	0.50	144	20	233	32	Yes
	C-T	59	40	0.80	74	18	461	44	Yes
	P-T	78	41	0.60	130	22	299	31	Yes
	P-24	88	41	0.55	160	22	253	19	Yes
9 (M, 58)	C-24	81	45	0.40	202	18	148	27	No
	C-T	76	42	0.40	190	21	157	29	No
	P-T	66	42	0.40	165	27	167	25	No
	P-24	65	37	0.45	144	30	210	27	No
10 (F, 27)	C-24	65	35	0.50	130	20	248	12	No
	C-T	77	30	1.0	77	17	598	21	No
	P-T	74	27	0.75	99	22	427	31	No
	P-24	70	31	0.69	117	22	319	32	No
11 (F, 65)	C-24	65	45	0.80	81	15	449	22	No
	C-T	78	52	0.75	104	14	392	26	No
	P-T	64	48	0.70	91	16	375	25	No
	P-24	66	49	0.65	102	17	336	25	No
12 (M, 4)	C-24	48	34	1.0	48	5	622	16	No
	C-T	56	46	1.0	56	5	600	27	No
	P-T	146	23	1.0	146	8	538	30	No
	P-24	159	27	0.90	177	6	449	32	No
13 (F, 8)	C-24	62	38	1.0	62	10	604	30	No
	C-T	54	36	1.0	54	10	614	28	No
	P-T	63	34	1.0	63	9	608	36	No
	P-24	60	31	0.90	67	12	543	37	No
14 (F, 46)	C-24	79	37	0.90	88	29	516	24	No
	C-T	76	37	0.90	84	22	519	31	No
	P-T	225	29	0.90	250	21	380	34	No
	P-24	97	31	0.50	194	26	221	35	No

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*C-24 = settings on CMV 24 h before transition; C-T = settings on CMV at transition; P-T= settings on PCV at transition; P-24 = settings 24 h after transition.

†Mean (SD) P_{aO_2} P-T – P_{aO_2} C-T = 25.4 (45); $p = 0.055$. Mean (SD) P_{aO_2} P-24 – P_{aO_2} C-T = 22 (37); $p = 0.046$.

‡Mean (SD) P_{aCO_2} P-T – P_{aCO_2} C-T = 8.2 (15); $p = 0.058$. Mean (SD) P_{aCO_2} P-24 – P_{aCO_2} C-T = 7.1 (14.1); $p = 0.08$.

§Mean (SD) F_{IO_2} P-T – F_{IO_2} C-T = 4 (8.1); $p = 0.086$. Mean (SD) F_{IO_2} P-24 – F_{IO_2} C-T = 15 (14.4); $p = 0.002$.

#Mean (SD) P_{aO_2}/F_{IO_2} P-T – P_{aO_2}/F_{IO_2} C-T = 32.7 (50.8); $p = 0.032$. Mean (SD) P_{aO_2}/F_{IO_2} P-24 – P_{aO_2}/F_{IO_2} C-T = 50 (57.2); $p = 0.006$.

¶Mean (SD) C_{dyn} P-T – C_{dyn} C-T = 2.2 (2.3); $p = 0.002$. Mean (SD) C_{dyn} P-24 – C_{dyn} C-T = 3.2 (3.4); $p = 0.004$.

**Mean (SD) $P_{(A-a)O_2}$ P-T – $P_{(A-a)O_2}$ C-T = 43.6 (66.5); $p = 0.03$. Mean (SD) $P_{(A-a)O_2}$ P-24 – $P_{(A-a)O_2}$ C-T = 118 (116); $p = 0.002$.

††Mean (SD) P_{aw} P-T – P_{aw} C-T = 1.91 (10.8); $p = 0.33$. Mean (SD) P_{aw} P-24 – P_{aw} C-T = 1.94 (9.4); $p = 0.46$.

Table 3. Summary of Changes Occurring in 14 ARDS Patients between Conventional Mechanical Ventilation at Transition (C-T) and Pressure-Control Ventilation at 24 Hours after Transition (P-24)

Variable	Mean C-T*	Mean P-24	Mean (SD) of Difference
$P_{aw\text{-}peak}$ (cm H_2O)	64	51	13.3 (12.5)
PEEP (cm H_2O)	12	10	2.7 (5.2)
F_{IO_2}	0.78	0.63	0.147 (14.4)
$\dot{V}_E$ (L)	15	12	3.1 (2.4)
C_{dyn} (mL/cm H_2O)	16	19	3.2 (3.4)
$P_{(A-a)O_2}$ (torr)	433	315	118 (116)
$\bar{P}_{aw}$ (cm H_2O)	29	31	1.9 (9.4)
Inspiratory time (%)	44	47	3.4 (19.2)

*The decelerating ramp waveform was used during CMV.

the ability to ventilate at lower $P_{aw\text{-}peak}$. This improved oxygenation probably occurs as a result of reduction in shunting, alveolar recruitment, decreased dead-space ventilation. These clinical improvements probably occur secondary to increased mean airway pressure, increased inspiratory time, and increased functional residual capacity (FRC).

Clinicians should be cautious about several aspects of PCV application. Because $P_{aw\text{-}peak}$ is constant, flowrate and V_T vary with changes in patient resistance and compliance. Alarms considered to be appropriate indicators of potential patient compromise with CMV are not helpful with PCV. For example, a high-pressure alarm used in CMV is usually considered to provide a first-line alert for conditions such as airway obstruction and barotrauma. During PCV, this alarm does not alert the clinician to such events. This is potentially dangerous in patients managed with paralyzing agents or those unable to actively exceed the threshold of the high pressure alarm-limit setting (eg, unable to cough). The potential exists for airway obstruction without activation of the high-pressure alarm.

The $P_{aw\text{-}peak}$ with which the patient is ventilated during PCV is the sum of PEEP and set $P_{aw\text{-}peak}$. As PEEP is adjusted, the applied $P_{aw\text{-}peak}$ increases or decreases by the same amount. Conditions that we transiently observed included a decrease in V_T as PEEP increased (even though applied $P_{aw\text{-}peak}$ increased by the same absolute value as the PEEP adjustment). This may have been the result of being very high on the patient's pressure-volume curve by virtue of the presence of auto-PEEP. This effect can decrease compliance, secondary to air trapping, resulting in a decrease in the exhaled V_T measured. Conversely in this example, a decrease in PEEP increased V_T. Secondly, excessive lengthening of t_i can decrease exhaled V_T because of inadequate expiratory time (resulting in air trapping). PEEP reduction in this situation restored V_T probably because (1) air trapping existed and (2) the resultant PEEP adjustment established ventilation at a lower level on the pressure-volume curve.

Decreasing PEEP while maintaining $P_{aw\text{-}peak}$ effectively increases V_T because of the increase in the pressure difference of the inspiratory pressure curve. Conversely, if $P_{aw\text{-}peak}$ is held constant and PEEP is increased, V_T decreases secondary to a decreased pressure difference during inspiration.

The length of inspiration affects V_T, and, thus, t_i must be long enough for full equilibration to occur. With long t_i at high rates, air-trapping and auto-PEEP can develop. Barotrauma, systemic hypovolemia, and hemodynamic embarrassment have been attributed to the presence of auto-PEEP.[11,16-19] Auto-PEEP should be monitored according to appropriate reported methods[11-14,18] and by serial chest radiographs to identify hyperinflation and by evaluation of hemodynamic variables. The low-to-nonexistent auto-PEEP in our patients probably reflected the hastened lung emptying seen in patients with low lung compliance[20] and the conservative t_i and low breathing rates in most of our patients.

Although only 4 of our patients (#1, 8, 10, and 14) were monitored with Swan-Ganz catheters and only 2 of those (#1 and 10) were ventilated with markedly inverse I:E (t_i% 75 and 78, respectively), no evidence of important hemodynamic embarrassment was seen. None of the 14 patients experienced a compromise in blood pressure. Although our data are not complete enough to establish absence of hemodynamic embarrassment, we believe that use of conservative I:E and maintenance of adequate blood volume are important.

Algorithms have been used to guide the ventilation of neonates[21] and the utilization of blood-gas analysis[22] as well as other clinical procedures.[23] Our algorithm (Fig. 1) has aided our staff by providing guidelines for the consistent management of P_{aO_2}, P_{aCO_2}, and V_T during the preweaning stages of PCV.

Summary

I believe that patients suffering from acute respiratory failure associated with ARDS who require high $P_{aw\text{-}peak}$, PEEP, and $\dot{V}_E$ during CMV can be adequately ventilated with PCV. Using PCV, my colleagues and I observed significant improvement in $P_{aw\text{-}peak}$, C_{dyn}, $\dot{V}_E$, and $P_{(A-a)O_2}$, without significant changes in P_{aO_2}, P_{aO_2}/F_{IO_2}, and f. The extreme inverse I-E ratios reported by previous investigators[4-6] were not found to be necessary in all cases. The improvements that we observed occurred with inspiration maintained at 50% or less of the breath cycle (I:E $\leq$ 1:1) in 8 of 14 patients (57%).

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Computers in Pulmonary Medicine: Ten Years Ago, Today, and Tomorrow

A little more than a decade ago, it was the sense of this journal's editors that computers were about to become important tools in pulmonary medicine. Only a few papers about computers had been published in these pages, but such material was appearing with increasing frequency in other journals, and we expected more and more computer-related papers to cross our editorial desks. Therefore, feeling ignorant about computers in general, and particularly in medicine, and assuming that many of RESPIRATORY CARE's readers were in the same fix, we pulled together what material we could find on the subject, publishing it in a special issue, "Computers in Pulmonary Medicine," in July 1982.[1]

Today, ten years later, computers are a familiar presence in labs, ICUs, nursing stations, and libraries; and in some medical centers you will find a Department of Informatics (a word derived from the Russian language[2]). In a decade, the unfamiliar and daunting has become accepted and almost commonplace. The computer revolution has been one of the fastest moving in history. In retrospect, it may turn out some day that computers have made as much difference in our lives as have electricity, the telephone, the automobile, aircraft, radio, and television.

To mark the 10th anniversary of our 1982 special issue on computers, in this January 1993 issue we offer five papers that help define computers in pulmonary medicine today—and their projected impact in the future. In 1982, six papers reported the use of computers in pulmonary function testing and blood gas/pH analysis, which were early applications. Today, the frontiers reflected here are essentially two: (1) the clinical and management use of computers, to which two papers in this issue are devoted,[3,4] and (2) computers in the ICU environment, particularly that of the future, explored in three other papers.[5-7]

As suggested by these papers' titles (see references below), the future of computers in pulmonary medicine promises to be anything but dull.

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Computerized Management of Respiratory Care

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I. Introduction
II. Computer Applications & Reports
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  B. 24-Hour Management Report
  C. Medical & Management Corrective/Alert Report
  D. Productivity & Billing Report
    1. Productivity Applications
    2. Billing
    3. Editing
III. Summary

Introduction

From the time when respiratory therapy became an organized discipline (about 1948) through the 1970s, respiratory care departments were seen by hospital management as revenue centers. But this changed in the 1980s, and respiratory care services, like many other services, came to be viewed as cost centers. This occurred in association with the 1982 Blue Cross and Blue Shield Medical Necessity Guidelines,[1] the advent of the Diagnosis Related Group (DRG) system in 1983,[2] and the 1988 Medicare Catastrophic Coverage Act.[3] These events changed management principles and practices in hospitals. Developments in computers at roughly the same time made possible entirely new ways of monitoring, recording, and interpreting the respiratory care provided to patients and the productivity and cost-benefit ratios of individual therapists and respiratory care departments. This paper describes how respiratory care management has been computerized at LDS Hospital, a 520-bed trauma center in Salt Lake City, Utah, associated with the University of Utah School of Medicine.

LDS Hospital is a leader in the development of computer applications in medicine. A highly developed Hospital Information System (HIS), known as HELP (an acronym for Health Evaluation through Logical Processing), integrates all patient information.[4] A Tandem "nonstop" TNS II computer system connects more than 800 terminals and printers, forming a networked comprehensive data-management system. This system is highly reliable and, due to its redundant processing and storage of data, has only 0.2% downtime.[5]

The computerization of respiratory care documentation and the utilization of the HELP system add to the comprehensive database developed on each patient. The database includes information from all medical and nursing departments, as well as financial data. The data are integrated in such a way that inputting, accessing, reporting, reviewing, and editing are available at either bedside or care-unit remote terminals.

The Department of Respiratory Care has utilized the Tandem Computer System in its comprehensive form since 1983, the end product of the master's studies project of a Medical Informatics graduate student.[5]

Computer Applications and Reports

Figure 1 shows the integrated network of information processed from the respiratory care doc-

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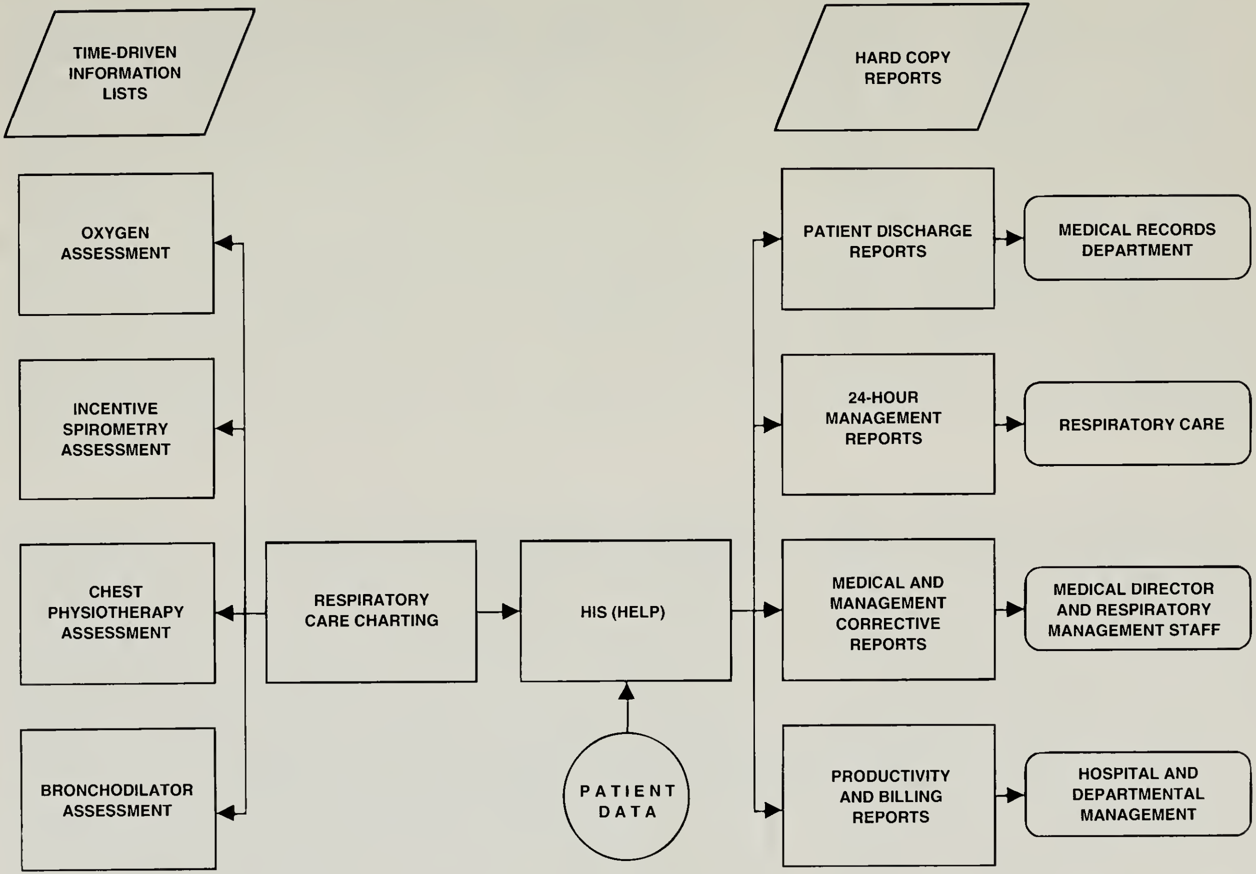
TIME-DRIVEN INFORMATION LISTS
OXYGEN ASSESSMENT
INCENTIVE SPIROMETRY ASSESSMENT
CHEST PHYSIOTHERAPY ASSESSMENT
BRONCHODILATOR ASSESSMENT
RESPIRATORY CARE CHARTING
HIS (HELP)
PATIENT DATA
HARD COPY REPORTS
PATIENT DISCHARGE REPORTS
24-HOUR MANAGEMENT REPORTS
MEDICAL AND MANAGEMENT CORRECTIVE REPORTS
PRODUCTIVITY AND BILLING REPORTS
MEDICAL RECORDS DEPARTMENT
RESPIRATORY CARE
MEDICAL DIRECTOR AND RESPIRATORY MANAGEMENT STAFF
HOSPITAL AND DEPARTMENTAL MANAGEMENT

Fig. 1. The integrated network of information depicts the information provided by computer charting at LDS Hospital. Data from respiratory care charting go both to the time-driven information lists on the left and to the hard-copy reports on the right. The arrows indicate the pathway of information and the associated process performed and reported as an automatic byproduct of computer charting.

umentation. The arrows indicate the pathway of information and the associated processes performed and reported. Four reports are generated as a result of therapist documentation: (1) Patient Discharge Report, (2) 24-Hour Management Report, (3) Medical and Management Corrective Action Report, and (4) Productivity Report and Patient Billing.

In addition, four time-driven lists of information are printed daily from automated computer searches: (1) Oxygen Assessment, (2) Incentive Spirometry Assessment, (3) Chest Physiotherapy Assessment, and (4) Bronchodilator Assessment. The data from each patient receiving one or more of these modalities of care are comprehensively reviewed every 3 days to objectively document the results of each specific modality. In this way, criteria-based respiratory care is delivered and documented concurrently.

Patient Discharge Report

One of the responsibilities of providing patient care is recordkeeping. Standards of the Joint Commission for Accreditation of Healthcare Organizations (JCAHO) require that "reports of all respiratory care services provided to the patient shall be made part of the patient's medical record."[6] Recordkeeping systems depend primarily on the information collected at the bedside. This information is the result of the therapist's interaction with the patient. Reports of physical examination and the patient's response to each modality of care comprise the data for documentation. Traditional respiratory care documentation has been handwritten in the patient's medical record, using specific forms, notes, assessments, and flow sheets. Historically, each section is organized chronologically and consumes considerable space in the medical record, making it cumbersome to handle; however, this documentation is mandated by medical practice and legal requirement.

The use of manual records in carrying out a systematic program of medical and nursing intervention, health monitoring, and scheduled treatment is difficult and time-consuming. The nonmedical functions of recordkeeping—such as billing, audits, meeting government requirements, and satisfying legal considerations—have been the driving force behind the introduction of computers to improve medical-information management. Whereas previously hospital computer systems were used primarily to bill patients, it has now become necessary to computerize and integrate medical-record information to improve the timeliness and completeness of the medical record.

Patient records vary in quality and detail because from one third to one half of them are narrative in nature,[7] which makes data difficult to collect and process. Respiratory care charting is predominantly written in this narrative format, with the exception of ventilator-monitoring data, which are predominantly numeric in nature.

Computerized respiratory care information systems require a reporting vocabulary with a wide range of descriptions. To automate patient records, the records must be converted from a narrative format to the computer's predefined vocabulary (Fig. 2).

24-Hour Management Report

The 24-Hour Management Report lists the work charted for that period by each therapist (Fig. 3). It reports, in relative value units (RVUs), the total number of treatments performed by an individual therapist and the actual time spent performing each treatment. (RVUs are engineered time standards.) Thus, it is a record of individual productivity. Supervisors review the report to confirm that assigned procedures have been completed and to identify missed procedures so that appropriate corrective action can be taken. A department summary lists total procedures performed and the reasons for treatments not completed (Fig. 4). The individual and 24-hour summary reports provide management with data for long-term individual and department historical collection. These data are the basis for the individual pay period productivity that is reviewed at each supervisory meeting by all members of the management team (Fig. 5). This process has become invaluable in tracking individual productivity. These data are then used for individual comparison and become an essential data set for the employee's annual performance review (Fig. 6).

One advantage of using this type of reporting for long-term follow-up is that the 24-hour report compares the individual therapist's documentation of actual time spent performing each procedure to the

LDS Respiratory Care Department
Vocabulary for Computer Reporting

Procedure:
Pulmonary Exercise
Medication Nebulizer
IPPB
CPT
Oxygen
Nebulizer Monitoring
Intermittent Nebulizer
Assessment
Treatment not done

Reasons Care Not Done:
Not on unit
Asleep
Receiving other care
Discharged
Nauseated
Due to workload
Refused care

Oxygen Interface:
Cannula
Mask
Face tent
Partial rebreather

Position:
Sitting
Dangling
Trendelenburg
Supine
Prone
Left side
Right side
Trendelenburg/right
Trendelenburg/left
(Trendelenburg/prone)
Left/flat
Right/flat

Breath Sounds:
Normal (both lungs)
Slightly decreased
(Moderately decreased)
Markedly decreased
Wheezing
Absent
Rhonchi
Bronchial
Egophony
Fine crackles
Coarse crackles
Inspiratory stridor
Unchanged
Improved

Description:
Throughout inspiration
Initial inspiration
Mid-inspiration
End-inspiration
Throughout expiration
On forced expiration
R > L
L > R

Location:
Both lungs
Left lung
LUL
Left lingula
LLL
BLL
Right lung
RUL
RML
RLL
LUL apical
LUL posterior
LUL anterior
LLL apical
LLL posterior
LLL anterior
LLL lateral
RUL apical
RUL posterior
RUL anterior
RLL apical
RLL posterior
RLL anterior
RLL lateral

Percussion:
Mechanical
Manual
Passive

Inspiratory Flowrate:
Adequate
Not Adequate

Medications:
Atropine
Alupent
Bronkosol
Isuprel
Micronephrine
Mucomyst
Alcohol

Cough:
Frequent
Infrequent
When encouraged

Sputum:
Spontaneous
Suction
Induced
Sterile
No sputum obtained

Sputum Color:
Clear
Cloudy
Cloudy white
Yellow
Brown hemoptic

Sputum Consistency:
Thick
Thin
Frothy
Tenacious
Mucus plugs
Viscid

Equipment:
Changed
Not Changed

Patient Condition:
Alert
Confused
Restless
Apprehensive
Fatigued
Quiet
Semiconscious
Comatose
Calm
Combative
Medicated

Patient Attitude:
Cooperative
Uncooperative
Not responding

Comments:
Treatment tolerated well
Patient needs encouragement
Poor effort
Doesn't work on own
Ambulatory
RN Notified
Febrile
Nauseated
Cyanotic

Fig. 2. Computerized respiratory care reporting vocabulary.

procedure's RVUs. This is an important process because RVUs like other time standards are engineered by the stopwatch observation method and are accurate at only one point in time. The time-standard methodology, though accurate, is not patient-specific or dynamic, whereas respiratory care is a dynamic practice, and the times taken to perform the same procedure on two different patients (or on the same patient on different occasions) may differ substantially. Time standards should ideally be re-evaluated annually. This is an expensive and labor-intensive process that we believe is universally unpopular. If, over time, the acuteness of the patient population's medical condition has changed or the efficiency of procedure delivery has improved, the actual recorded time probably will have changed. For example, from the RVUs recorded in one 24-hour period (Fig. 3), it is seen that although 417.19 standard RVUs were recorded, the actual recorded performance time was 300 minutes. This provides management with the necessary data to request a re-evaluation of or an adjustment in the time standards or productivity index.

A second advantage of the 24-hour report is that it is a 'smart' report that records the turnaround time, in minutes, between when a treatment was given and when it was documented. This information is invaluable relative to the timeliness of data entry. Many medical decisions at our institution are based on data-driven protocols. In the management of critically ill patients, the timeliness of information for decision making is critical to the

LDS RESPIRATORY CARE: THERAPIST REPORT 17 AUGUST 1984

Therapist #_____ Aug 17, 1984 00:00-Aug 17, 1984 23:59

Date	Time	Room	Patient	Entry	Treatment	RVUs	No. of Tx	Tx Time	TA	CT
08-17	12:00	E509	A	15:00	Med Neb	23.24	1	30	154	4.5
08-17	12:00	E509	A	14:17	Neb Monit					
					HPN Equip Setup	20.51				
					On O_2					
08-17	08:00	E509	A	11:46	Med Neb	23.24	1	20	209	3.5
08-17	07:30	E509	A	14:21	Neb Monit	16.73	7	10	402	1.5
08-17	07:30	E509	A	14:15	O_2					
08-17	07:00	E510	B	14:35	Neb Monit	19.12	8	10	419	1.5
					On O_2					
08-17	13:45	W623	C	14:35	Pulmo Exercise	15.04	1	20	33	3.5
					Med Neb	23.24				
08-17	09:30	W623	C	14:32	Pulmo Exercise	15.04	1	20	294	2.5
					Med Neb	23.24				
08-17	13:30	DSCH	B	14:57	Med Neb					
					not done					
08-17	11:40	DSCH	D	14:35	Med Neb					
					not done off unit					
08-17	07:40	DSCH	D	11:44	Med Neb	23.24	1	20	225	1.5
08-17	07:00	E514	E	14:12	O_2					
08-17	12:30	E512	F	14:45	IPPB	27.34	1	35	224	4.5
					CPT	24.61				
08-17	10:30	E512	F	14:45	IPPB	27.34	1	35	224	4.5
					CPT	24.61				
08-17	08:30	E512	F	14:41	IPPB	27.34	1	20	355	4.5
08-17	07:30	W629	G	14:23	Neb Monit	19.12	8	10	405	2.5
					On O_2					
08-17	07:30	E515	H	14:27	Neb Monit	17.12	8	10	408	1.5
08-17	07:15	W538	I	14:29	Med Neb	23.24	1	20	416	2.5

Total RVU:	417.19
Total Length of Procedures:	300 min
Average turnaround time:	277.2 min
Total computer time:	46.0 min
Average computer time:	2.6 min

TA = turnaround time; CT = computer time; Tx time = treatment time. All time in minutes.

Fig. 3. Therapist 24-Hour Management Report. This compares total standard relative value units (RVUs) to actual time spent in therapy by the therapist. This report also shows the average computer turnaround of 277 minutes in 1984.

LDS RESPIRATORY CARE DEPARTMENT REPORT 17 AUG 1984
24-Hour Management Report

RUN AT 13:31 18 AUG 84
RUN TIME: 24 MIN

SHIFT TOTALS

Average Points	Points	Entries	Average Entries
11.8	319.3	446	14.5
Average RVUs	RVUs	Entry Time	Average Time
354.79	9579.43	796	1.8
RVU/DUR	Therapist DUR	Average Turnaround Time	
1.36	7021	100.7	
Therapists	Charges	% RVUs Completed	% Charges Completed
27	8014.76	97.6	98.4

RX Completed	**Total**	**Initial**	**RVUs**	**Charges**
Pulmonary Exercise	87	14	1538.08	814.70
Medicated Nebulizer	85	2	2008.20	941.90
IPPB	18	1	508.52	204.52
CPT	53	3	1353.53	1066.00
Intermittent Nebulizer	1	1	35.54	14.00
Assessment	38	17	935.18	176.70
O_2 (Hrs)	1800		1206.00	3600.00
Nebulizer Monitoring (Hrs)	313		748.07	287.96
Suction	2		27.34	13.00
Hyperbaric RX (Min)	150		256.50	201.00
[Interhospital Transport (Min)]	60		60.00	66.00
Medical Nebulizer In-Line [Intrahospital Transport (Min)]	240		240.00	2264.00
Ultrasonic Nebulizer Equipment-Setup	3		41.01	30.00
Thoracic Demo	1		61.52	35.00
O_2 Equipment-Setup	11		150.37	110.00
Standby (Min)	20		20.00	00.00
			9579.43	8014.76

Reasons Treatments Not Done				
Not on Unit	1		23.24	10.90
Asleep	1		15.04	7.90
Receiving Other Care	1		15.04	7.90
Nauseated	1		24.61	20.00
Due to Workload	3		45.12	23.70
Refused Care	2		30.08	15.80
Advised Not to Give	1		24.61	20.00
Unable to Complete	1		15.04	7.90
Other	2		38.28	18.80
	13		231.06	132.90

Fig. 4. Respiratory Care Department 24-Hour Management Report.

outcome of the decision. It is expected that data will be documented within 30 minutes of the completion of care. The average turnaround time was 270 minutes in 1984 (Fig. 3), whereas in 1992 it had dropped to a mean (SD) of 35.14 (0.84) minutes (Fig. 7).

Lastly, the 24-hour report documents the time spent in the act of documentation. We feel that this

LDS RESPIRATORY CARE DEPARTMENT
PRODUCTIVITY
OCT 1992 – Pay Period 22

Name	Num	11	12	13	14	15	16	17	18	19	20	21	22	23	24	RVU Totals	Prod Hrs	% Prod	Total Hrs	OT Hrs
						165	485	284			191	461	324	180	135	2226	39.25	95	55.25	
																0			.00	
																0			.00	
		499													452	950	16.00	99	16.00	
									641							641	7.00	153	7.00	
			391	344	546	669	115			527	587	636	699			4513	73.50	102	81.50	1.50
		932	1012					898	841	1004					1104	5791	72.25	134	72.25	
		128	109										655	50	201	1143	15.25	125	63.25	
																0			.00	
				962	904			918	958					996	644	5383	72.50	124	72.50	
	Totals:	1559	1512	1306	1450	834	600	2100	2440	1531	778	1097	1678	1226	2536	20647	296	116	368	2
	Adult:	13177	14219	14850	15632	14528	17213	17972	16443	17655	15967	15664	15345	15314	17426	221405	296	—		
	Home Care:	0	740	768	924	386	548	454	204	302	858	808	264	886	0	7142				
	HBO:	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0				
	NBICU:	904	1775	1893	1767	1697	1411	1317	941	742	1095	888	642	426	285	15783				

Total FTEs: 4.60 Total Productive FTEs: 3.70 Average RVUs: 1475

NON-PRODUCTIVE HOURS

	Hours	FTEs
Administrative	8.0	.10
Supervisors	48.00	.60
$ECCO_2R$	.00	.00
Inservice	.00	.00
Orientation	16.00	.20
Smoke Stoppers	.00	.00
Secretarial	.00	.00
Total	72.00	.90
HBO	.00	.00
NON-PRODUCTIVE PERCENTAGE		.08

Fig. 5. Pay-Period Individual Productivity Report. This also summarizes the nonproductive activities of the department in the pay period.

is important to dispel the idea that computer documentation is as time-consuming as manual documentation. An average of 2.6 minutes was spent in documentation in 1984 (Fig. 3), compared to 1.37 (0.14) minutes in 1992 (Fig. 7).

LDS Respiratory Care Department
Individual Productivity

		Pay Period							
Name	Average	1	2	3	4	5	6	7	8
1	91%	110	77	90	69	71	108	110	
2	99%	102				96			
3	91%	97	78	54	84	93	117	107	99
4	109%	105	104	115	107	113	108	103	118
5	139%	134	180	117	195	132	110	108	
6	120%	146	97	186	126	135	80	74	
7	137%	157	122	136	148	151	115	127	
8	99%	107	95	103	112	96	77	102	
9	121%	133	120	119	121	129	110	114	
10	95%	110	78	101	99	98		83	

Fig. 6. Eight-pay-period mean individual productivity for the department.

Medical and Management Corrective/Alert Report

The Management Corrective Action Report (Fig. 8) is used for monitoring errors in the documentation of respiratory care procedures. An example of a management corrective action is an overcharge resulting from double-charting. For example, if oxygen administration is documented for more than 24 hours in a single day, a corrective action alert is printed so that the charting and billing can be corrected. Medical alerting is discussed in another paper in this issue of RESPIRATORY CARE.[8]

LDS Respiratory Care Department
Charting Summary

Turn Around	Elapsed Minutes
Time, mean (SD)	35.14 (0.84)
Time/Entry	1.37 (0.14)
RVU/Therapist	551 (2.0)
Total Entries	19973

Fig 7. Respiratory Care Department data for the period 09/29-10/27, 1990.

Productivity and Billing Report

Productivity Applications. By convention, productivity is defined as the ratio of input to output, expressed as a percentage. The ratio of individual hours paid to work performed is an indicator of productivity. Respiratory care is a relative newcomer (1981) to the application of this methodology in staff management.[9] Productivity management is still not uniformly applied as a management practice in respiratory care departments nationally; and even in those departments where it is practiced, it is not usually attached to the annual performance review process. The reason for the lack of application may be found in the diversity of the scope of practice in respiratory care and the lack of validated time standards by procedure.

The American Association for Respiratory Care has made significant contributions in this area by publishing the "Respiratory Therapy Uniform Reporting Manual" in 1981[9] and 1984.[10] This manual explains the basics of productivity management, including a list of procedures and time standards, and provided the framework for the productivity system developed by the Department of Respiratory Care at LDS Hospital. Then, in 1983, the Department, with the aid of the Intermountain Health Care (IHC) Industrial Engineering Section, developed a list of procedures performed in the Department, and, with stopwatch and observation, validated the actual time spent performing each procedure (see Appendix).

Billing. The charting of completed procedures automatically generates the patient's hospital charge. Billed items selected during the clinical charting determine what account numbers are stored. Billing is an automatic byproduct of computer charting of the procedure. Multiple billable items are embedded throughout the charting so that an item is billed only if it is charted. The therapist never has to prepare billing records; they are determined by the charting. This method provides direct correlation between the charted procedure and the patient's bill. Billing accuracy depends on the therapist's charting accuracy, and mistakes occasionally occur, such as charting on the wrong patient or charting the same procedure twice. These

LDS Respiratory Care Department
Corrective Action Report

Reviews 24 Hours of Information from
23:59 OCT 05, 1992

ROOM	PATIENT	CPAP TOTAL	VENT TOTAL	USN TOTAL	HPN TOTAL	OTHER TOTAL	TOTAL O_2 HRS.
0001	Patient #1 > 24 Total Hrs. HPN Interrupted or DC'd	0	10	0	18	0	28
0002	Patient #2 > 24 Total Vent Hrs.	0	26	0	0	0	26
0003	Patient #3 > 24 Total O_2 Hrs.	0	14	0	17	0	31
0004	Patient #4 HPN Interrupted or DC'd						
0005	Patient #5	0	26	0	5	0	31
0006	Patient #6 HPN Interrupted or DC'd						
0007	Patient #7 > 24 Hrs. O_2	0	0	0	0	28	28
0008	Patient #8 O_2 Interrupted or DC'd						
0009	Patient #9 USN Interrupted or DC'd						
0010	Patient #10	0	28	0	0	0	28
0011	Patient #11 Initial O_2 Equip setup not documented O_2 first documented 10/05.10:00 Therapist						
0012	Patient #12 > 24 Hr. USN	0	30	0	0	0	30

Fig. 8. 24-Hour Corrective Action Report.

errors can be found easily by the therapist when he/she reviews the charting—or by supervisors when they review the 24-hour management report. Errors can easily be corrected by supervisory personnel. Billing accuracy is not merely a concern to the hospital and patient, but it also determines respiratory care productivity, which is used to establish staffing requirements for the department.

Editing. Both billing and clinical charting can be edited. If a mistake was made in charting, a form is filled out by the therapist and given to the shift supervisor. The supervisor then uses programs available only on the department terminal to credit billing overcharges or to delete duplicate charting. One systems program is used to edit clinical charting, and another systems program is used to credit billing overcharges. In order to maintain control of patient-billing functions, only specific supervisors can access the editing and billing crediting programs.

Summary

Respiratory care as an organized discipline is only about 45 years old, and the management of this dynamic allied health profession has usually been characterized by a demand-for-service mentality. As pressure continues to control costs, those departments that maximize quality patient care cost-effectively with thoroughly documented outcomes are in a better position to compete for future resources. The practice of respiratory care is changing as is the practice of medical care in general. Accountability for resource consumption and the quality of the product delivered are essential elements in the delivery of respiratory modalities. We have developed and implemented a comprehensive patient-data-based approach to the management of respiratory care. The essential elements of this approach are (1) relative-value-unit procedure base; (2) individual, shift, and department productivity that is attached to the annual performance review process; (3) management reporting on a 24-hour basis, with biweekly review at the management level; (4) development and implementation of a comprehensive patient-data-documentation system that permits automatic patient billing and 100% data review for quality-assurance documentation; (5) the development of a medical alerting system that alerts the Medical Director and Respiratory Care staff to potentially harmful events that, if untreated, may result in increased morbidity or mortality; and (6) the development of concurrent and retrospective tools for patient-outcomes research. These functions are supported by an active Medical Informatics Department that is nationally recognized in medical computing and logic application.

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APPENDIX

LDS RESPIRATORY CARE

Relative Value Units (RVUs) by Procedure

DESCRIPTION	RVUs
Assessment Patient-Chest Physiotherapy	49.57
Assessment Patient-Incentive Spirometry	24.61
Assessment Patient-Oxygen	24.61
Ventilator/CPAP Equipment at Bedside, Hour	0.00
S/U Pressure Monitor (PK,PEEP,MA)	13.67
Ultrasonic Nebulizer: Patient Care Monitor, Hour	2.39
CPAP Continuous-New Born IBU, Hour	13.67
Ventilator-New Born IBU, Hour	13.67
Special Procedure: Trauma (Charge/minute)	1.00
Special Procedure: CPR LSU (Charge/minute)	1.00
Manual Ventilator Equipment/Supplies	0.00
Set Up Equipment, Apnea Monitor	21.87
Set Up End Tidal CO_2	13.67
Delivery Call-Charge/minute	1.00
CPT Equipment/Supplies	0.00
CPT Instruction/Education	16.40
Pulmonary Exercise Instruction/Education	16.40
Medications Nebulizer Equipment/Supplies	0.00
Medications Nebulizer Instruction/Education	16.40
IPPB Equipment/Supplies	0.00
IPPB Instruction/Education	16.40
Transtracheal Assistance/Education	70.00
CRS Therapist Service	45.00
Therapist Assistance/Minute	1.00
Monitoring Devices	
Late Charge or Adjustment	
Assessment Patient-Initial	24.61
Assessment Patient-Follow-Up	24.61
Extubation	13.67
Therapist Service	123.02
Patient Education & Training	43.74
Airway Suction	13.67
Airway Tracheal Tube Change	34.18
Airway Tracheal Lavage (Instill)	34.18
Airway Tube Care (Tracheal Care, Tie . . .)	13.67
Chest Physiotherapy Treatment	24.61
Chest Physiotherapy Infant (Less Than 2 Years)	12.30
Chest Physiotherapy Treatment-Initial and Education	41.01
IPPB Treatment	27.34
IPPB-Initial Treatment and Education	43.74
Set Up Equipment-Room Humidifier	6.84
Medications Nebulizer Treatment	23.24
Medications Nebulizer Treatment In-Line (Meds)	8.20
Medications Nebulizer-Initial Treatment and Education	39.64
Nebulizer-A-Patient Care Monitor per Hour	2.39
Set Up Equipment, Ultrasonic Nebulizer	13.67
Set Up Equipment, Nebulizer (HPN)	20.51
O_2 Hood-A-Patient Care Monitor Hb per Hour	6.84
Set Up Equipment, Hood (Monitoring)	20.51
Portable O_2 System Service	13.67
Set Up Equipment, Oxygen	13.67
Pulmonary Exercise Treatment	15.04
Pulmonary Exercise-Initial Treatment and Education	31.44
Self-Propelled Nebulizer Treatment (Inhaler)	13.67
Self-Propelled Nebulizer Treatment-Initial and Education	30.07
Sputum Induction (NT Suction)	27.34
Ventilator-A-Patient Care Monitor per Hour	13.67
Set Up Equipment, Ventilator (Time Cycled)	21.87
Set Up Equipment, CPAP Continuous	21.87
Set Up Equipment, Ventilator (Volume)	21.87
Supplies and Equipment	13.67
Intermittent Nebulizer Treatment (USN HPN Treatment)	19.14
Medical Gas, O_2/hour	0.68
Medical Gas, Air/hour	0.68
Intermittent Nebulizer-Initial Treatment and Education	35.54
CPAP Continuous-A-Patient/hour	13.67
CPAP Treatment	41.01
CPAP Treatment, Initial and Education	57.41
Set Up Equipment, Oximetrix	34.18

DESCRIPTION	RVUs
Transcutaneous Monitor CO_2-A-Patient Care Monitor/hour	3.77
Set Up Equipment, Transcutaneous Monitor CO_2	13.67
Transcutaneous Monitor O_2-A-Patient Care Monitor/hour	3.77
Set Up Equipment, Transcutaneous Monitor O_2	13.67
V_D/V_T Ratio (Manual) 54.68	
Blood Gas Draw (Catheter)	5.47
Blood Gas Draw (Puncture)	15.04
Bedside Spirometry	27.34
PFY Respiratory Parameters, Bedside (VC) Peak Flow	45.11
Airway Intubation	20.51
Airway Intubation Assist	20.51
Set Up Equipment, Hyperbaric Chamber	20.51
Specialty Gas Treatment (CO_2, Helium)	1.00
Specialty Gas, Initial Treatment and Education	1.00
Special Procedure: CPR LSU Trauma (Charge/Minute)	1.00
Special Procedure: Hyperbaric Chamber, total body	1.00
Special Procedure:Transport Intrahospital	1.00
Intrahospital Transport	1.00
Pulse Oximeter/hour	2.00
Linde Walker Transport	30.00
Venti-Voice (Day)	0.00
Apnea Monitor/hour	4.00
Spirometer, Disposable Incentive	0.00
Nasal Trumpet Airway	0.00
Peak Flowmeter	0.00
Trachcare Catheter	0.00
Hyperbaric Chamber-ICU (Minute)	0.00
Suctioning-Education	216.00
Ventilator, Cuirass-Home	600.00
Ventilator-Home	480.00

DESCRIPTION	RVUs
Therapeutics-IPPB Education	144.00
Breathing Retraining Education	63.00
Therapeutics-Nebulizer Education	96.00
Therapeutics-Ultrasonic Nebulizer Education	108.00
Therapeutics-Chest Physiotherapy Education	168.00
Oxymizer-Education and Training	30.00
Therapeutics-Education Pulmo-Aide	144.00
O_2 Assessment-Saturation	138.00
O_2 Evaluation-Transcutaneous Monitor	147.00
Therapist Service O_2 Out of State-I	180.00
Therapist Service O_2 Out of Town-I	192.00
Therapist Service O_2 in Town-I	165.00
Therapist Service O_2 Airline	300.00
Therapist Service O_2 Out of State-A	204.00
Therapist Service O_2 Out of Town-A	225.00
Therapist Service O_2 in Town-A	141.00
End-tidal CO_2 Monitor/hour	7.50
Pressure Monitor (PK,PEEP,MA/hour)	3.75
Airway Monitor (VT,RR,MA/hour)	3.75
Oxymizer Cannula	0.00
Pulmo-Aide Unit	0.00
Linde Walker-Fill Only	15.00
1:1 Therapist Service	60.00
Therapeutics-Apnea Monitor	84.00
Transtracheal Kit	0.00
Small Volume Nebulizer-Disposable (Intl)-Take Home	0.00
Small Volume Nebulizer-Disposable (Only)-Take Home	0.00
Inspirease-Take Home	0.00
Accessory Kit, Small Volume Nebulizer-Take Home	0.00
Nasal Cannula 7 foot-Take Home	0.00
Nasal Cannula 24 foot-Take Home	0.00
Assessment Patient-Bronchodilator	20.00
Assessment Patient-Basic	12.00

Quality Assurance for Respiratory Care Services: A Computer-Assisted Program

C Gregory Elliott MD

I. Introduction
II. History & Development
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Introduction

The medical director of respiratory care is responsible for assuring that established policies are carried out, that the quality, safety, and appropriateness of respiratory care services are monitored and evaluated, and that appropriate actions based on findings are taken.[1] The usual methods for discharging these responsibilities are rounds, at the bedside or in conference, and medical audits. Rounds are intended to provide concurrent monitoring of patient-care activities, but they are of necessity limited to a small patient sample. Thus, important patient problems are missed. The medical audit is designed to provide more complete data, but the audit is limited because it is retrospective (not all data may be recorded or sought), time-consuming, and samples only a small fraction of the total database.

Because of these limitations, the Respiratory Care Department and the Department of Medical Informatics at LDS Hospital developed a computer-assisted program for the quality assurance of respiratory care services. This paper describes the development process and the current operational status of the system. It also provides examples of the system's performance, in contrast to previous methods for quality assurance.

History and Development

The Hospital Information System

A hospital information system (HIS) known as the HELP system has been continuously developed at LDS Hospital from 1975 to the present.[2-4] The HIS system runs on a Tandem "non-stop" computer, which provides a central database that integrates all patient data throughout the hospital ac-

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cording to real-time entries (Fig. 1). For example, respiratory therapists chart ventilator observations at bedside terminals, and laboratory technicians record arterial blood gas measurements into the same database from the laboratory. Automated instruments, nurses, respiratory therapists, laboratory technicians, ward clerks, and physicians enter data. The system provides more than 650 terminals and printers for data entry and retrieval. At least four terminals and one printer are located on each nursing division. Intensive care units (ICUs) are equipped with at least one terminal at each bedside.

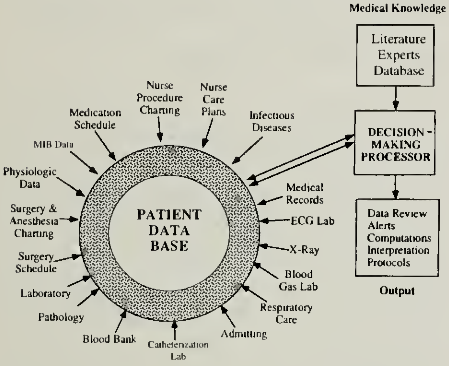

Fig. 1. The LDS Hospital HELP system features an integrated patient database that allows processing of the data for a variety of functions. Respiratory care procedures are charted into the patient database, and these data are reviewed every 24 hours for quality assurance of patient management ("alerts"). (Adapted from Reference 5, with permission.)

Once entered, the data are available for many medical and administrative activities, including quality assurance. The central patient database can be analyzed and processed to present specific information to a variety of health care personnel (data processing in Fig. 1), including physicians, nurses, and administrators. For example, physicians use the data processing function to perform a variety of physiologic computations for ICU data.[6] Physicians also use the data processing function to organize daily reports for clinical rounds activities on the wards or in the ICUs. Such reports present nursing observations (eg, vital signs, medications administered), as well as laboratory data (eg, serum electrolytes). The processing function also develops alerts based upon predetermined threshold values (eg, serum bicarbonate < 18 mEq/dL for metabolic acidosis).[7] Alert reports for the medical director of respiratory care are one important component of the LDS Hospital respiratory care quality assurance program.

Implementation of Respiratory Care Charting

By 1984, advanced data acquisition and processing existed in the ICUs and laboratories, but respiratory care charting was not part of the database, and processing of such data relied upon laborious retrospective chart audits. Furthermore, the quality assurance audits were focused, and they could not provide a broad overview of the quality and appropriateness of respiratory care services.

Illustrating the problem and the potential solution is the case of an elderly woman with a hip fracture who had received inappropriately prescribed oxygen therapy for 2 months. Because arterial blood gas data had been available in the database, it appeared possible to have examined these results at the time oxygen therapy was ordered. Such concurrent monitoring could identify and correct problems in a timely manner, rather than uncovering them months after the fact in a chart audit.

In order to provide surveillance of oxygen therapy and other respiratory care procedures, it was necessary to change respiratory care charting from a hand-entry to a computer-entry process. This transition was implemented and evaluated in 1984.[8] Respiratory therapists entered all procedures performed in all areas of the hospital. Approximately 18,000 entries were made daily (LDS Hospital is a 520-bed tertiary care center that provides a wide variety of services to medical and surgical patients). Evaluation showed that (1) therapists favored computer charting, (2) management procedures became more efficient, and (3) computer charting was more complete than handwritten entries (Fig. 2).

Once the respiratory care data were in the computer database, a quantum leap in respiratory care quality assurance activities became possible. The tedious and time-consuming process of chart audit was replaced by computer programs. Many of these programs are real-time, while others take minutes

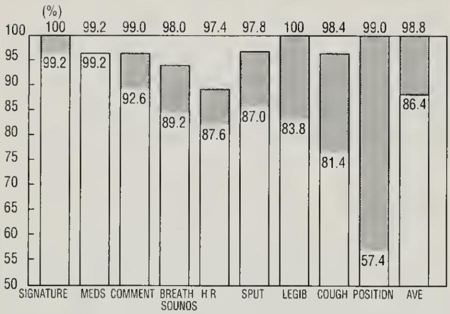

Fig. 2. Computer charting of respiratory care procedures is more complete than manual charting. Computerized records ▨ are also more descriptive and legible than manual ☐ charting. (From Reference 8, with permission.)

to run and allow review of all events under study rather than the usual limited sample. For example, inadequate oxygen therapy during intrahospital transport has been associated with significant complications, including hypotension and cardiac arrhythmias.[9] A respiratory care department may audit the compliance of its staff in documenting oxygen therapy while transporting patients in the hospital. We performed such an audit in 1982 and 1983, reviewing 104 charts. This process took a quality assurance coordinator more than 1 month to gather the data. Since the development of computer surveillance, all documentation of inspired oxygen fraction (F_{IO_2}) analyses for oxygen delivery is included in routine monthly screens of quality assurance. This process provides consistent and complete data without requiring repetitive, labor-intensive chart review. Furthermore, sampling bias is avoided because the system reviews every patient. In 1989, a monthly average of 663 F_{IO_2} analyses were reviewed by computer monitoring.

The computerized database also allows concurrent quality assurance of patient care. This process complements the usual word-of-mouth or incidental identification of immediate patient problems by providing systematic daily searches of the database for events such as cardiopulmonary arrests. Recently we demonstrated the power of these systematic hospital-wide searches.[10] We sought to identify patients who required reintubation within 24 hours of tracheal extubation. The quality assurance staff, utilizing a chart-review process, identified 8 events, whereas the computer surveillance system correctly identified 24 events within 24 hours of their occurrence. Although the time required for chart review was not measured, it was considerably longer than that taken by the computer search of the database.

These two processes—concurrent surveillance of patient care (medical director's alert) and regular monitoring of respiratory care procedures (policy and procedure screens)—were initiated in 1986 and 1987.[11]

Database Utilization for Quality Assurance

Medical Director's Alert Report

The medical director's alert report is a computer printout that identifies potentially dangerous situations throughout the hospital (Fig. 3). Every 24 hours, a program reviews the patient database. The purpose of this review is to identify dangerous situations in a timely manner. At present, the alert identifies 9 situations (Table 1). Each alert is based upon current medical knowledge (eg, safe versus unsafe tracheal tube cuff pressures[12]). The alert report 'filters' out problems that have been corrected (eg, arterial hypoxemia is not reported if a subsequent arterial blood gas documents a satisfactory arterial oxygen tension). At present, the computer program allows a time 'window' of 6 hours for the documentation of adequate oxygen therapy. In the future, time windows that are appropriate for specific areas of the hospital may be incorporated. Thus, the standard for documenting corrected hypoxemia may be 1 hour in the ICU and 3 hours on the medical ward.

The medical director's alert illustrates an important feature of HIS, an integrated database. The medical director's alert monitors observations made by respiratory therapists, arterial blood gas measurements, and microbiology studies (surveillance for nosocomial respiratory pathogens). The computer program can integrate these data for the alert report, and the program can incorporate additional data (eg, patient location in the hospital). Thus, the final alert report contains processed information that was entered by the respiratory therapist, the blood gas laboratory technician, and the ward clerk.

THERAPY ALERT

REVIEWS 24 HOURS FROM 06:00 NOV 19
06:05 NOV 20, 1992

E807	
11/19 19:00	Patient's IC has fallen more than 25% in 24 hours
W707	
11/19 11:19	ABGs have not been drawn within 6 hours of changing from a Vent, CPAP, or HPN to a cannula
E603	
11/20 05:45	Peak Pressure: 61 cm
11/20 05:45	$F_{IO_2} > 0.60$ for at least 16 hours
11/19 07:39	Cuff Pressure: 30 cm
E705	
11/19 02:55	Extubated Reintubated: 11/19 04:13
W711	
11/19 20:30	ABGs have not been drawn within 6 hours of changing from a Vent, CPAP, or HPN to a cannula
E605	
11/20 (NOC)	Cuff Pressure not checked in first 8 hours–74141
E711	
11/19 12:30	ABGs have not been drawn within 6 hours of changing from a Vent, CPAP, or HPN to a cannula
W807	
11/19 18:32	pH: 7.67 P_{CO_2}: 16.7 P_{O_2}: 77 S_{aO_2}: 96 F_{IO_2}: 21

Fig. 3. The Medical Director's alert report provides a review of respiratory care charting and arterial blood gases during the previous 24 hours.

These daily alerts lead to direct intervention by the medical director of respiratory care. Shortly after the alert system was activated, the alert report identified a seriously hypoxemic patient (Fig. 4). The alert presented a dangerously low arterial oxygen tension without evidence that oxygen therapy had been intensified, and with evidence that the hypoxemia had not been corrected. Furthermore, the high pH and low P_{CO_2} suggested that the hypoxemia was real—ie, the sample was not venous blood. The medical director located the patient on a surgical oncology floor. The patient had undergone a radical neck procedure and tracheotomy. The arterial blood gas had been ordered by a "cross covering" house officer who had been called to the operating room. Tracheal suctioning reversed the hypoxemia. In this example, the problem was identified at 0830 and corrected within the hour.

Table 1. Alert Conditions for the Medical Director of Respiratory Care*

Cardiopulmonary arrest
$P_{aO_2} < 55$ torr or $S_{aO_2} < 87\%$†
$F_{IO_2} \geq 0.6$ for more than 16 hours
PEEP > 10 cm H_2O
Tracheal-tube-cuff pressure > 27 cm H_2O
Airway temperature > 39°C
pH < 7.30 and $P_{aCO_2} > 50$ torr
pH > 7.54 and $P_{aCO_2} < 30$ torr
25% decrease of inspiratory capacity on incentive spirometry

*These conditions are identified from patient care data entered during the preceeding 24 hours. P_{aO_2} = oxygen pressure in arterial blood; P_{aCO_2} = carbon dioxide pressure in arterial blood; F_{IO_2} = inspired oxygen fraction; PEEP = positive end-expiratory pressure.

†Criteria for this alert include data integrated from the ABG laboratory and respiratory care charting. The integration of data reduces the number of false-positive alerts.

Date	Time	Location	Alert
2/1/88	0320	N481	P_{O_2} 43, F_{IO_2} 0.3 pH 7.71, P_{CO_2} 18
No ABG Follow-up in 6 hours, F_{IO_2} checked at 0.3-2/1/88, 0100.			

Fig. 4. An example of a medical director's alert.

Although such occurrences are rare, they illustrate the important 'safety-net' function of the medical director's alert. This safety net monitors all areas of the hospital for potentially dangerous situations that the patient's attending medical staff may have missed. Such a function may be particularly important for respiratory care departments and laboratories as more patients find themselves without coverage by medical and surgical physicians in training (residents).[13]

The medical director's alert also leads to corrective action through medical education or product modifications. For example, review of alerts triggered by high tracheal-tube-cuff pressures revealed that most high cuff pressures originated in the emergency room and the operating room. Education of the emergency room staff led to routine measurement of tracheal-tube-cuff pressure following emergency intubation. Review of anesthesia procedures revealed that tracheal tubes with undesirable low-volume, high-pressure cuffs were available for intubation. The incidence of high tracheal-tube-cuff pressures was sharply reduced by eliminating the purchase of tracheal tubes with low-volume, high-pressure cuffs.

Rate-Based Clinical Indicators (Policy and Procedure Screens)

In addition to the medical director's alert, the respiratory care quality assurance program utilizes the computer database to monitor 19 rate-based indicators of respiratory care procedures (see Table 2 for examples). These indicators are "process" indicators (ie, scientific data exist to link the care activities to patient outcomes). Because all data are charted in the computer, rate-based clinical indicators include all procedures performed and charted. As with the medical director's alert, the processing function of the HIS system provides the data without traditional chart reviews, which are time-consuming and expensive. Because of the ease of this process, the respiratory care quality assurance committee can review staff performance in all 19 areas monthly, a quality assurance activity that is not possible with traditional methods.

The respiratory care quality assurance committee meets every 3 months to review rate-based indicators. The committee initiates additional assessment if a rate-based indicator falls above or below predetermined thresholds. The thresholds were developed statistically (95% confidence interval) from analysis of monthly data obtained during the first year. Importantly, these data are developed with a reproducible method (program). This allows for continuous quality improvement (ie, the effects of educational programs or staffing changes on rate-based indicators of the quality of care can be measured). In order to achieve continuous quality improvement, the educational or staffing change should improve outcome, decrease cost, or both.[14]

Quality Assurance of the Database

"Garbage in results in garbage out" aptly describes a potential pitfall of computerized quality assurance programs. The integrated medical database permits unprecedented surveillance of respiratory care, but how does the surveyor know that the database is accurate? Ultimately, the surveyor must study the accuracy of the database.

Table 2. Examples of Rate-Based Clinical Indicators for Respiratory Care*

	Sample of Monthly Data	Upper or Lower Limit†
Ventilator care		
F_{IO_2} analyzed q 12 hours	96.6	86.8 (L)
Circuit changed qod	97.1	81.8 (L)
Suction catheter changed qd	98.5	82.6 (L)
Bronchodilator aerosols		
Heart rate > 130 beats/min	3.0	3.4 (U)
Adverse reaction to medication	0.4	0.7 (U)
Tracheal tubes		
Cuff pressure measured q shift	96.9	88.9 (L)
Cuff pressure > 27 cm H_2O	2.5	6.0 (U)
Extubation		
Respiratory parameters ≤ 3 hrs before extubation	88.7	78.3 (L)

*All data are % of observations that meet the departmental standard of care or that identify an adverse situation.

†Upper (U) and lower (L) limits represent 95% confidence interval for observations of these clinical indicators during the first 12 months of data analysis. An observation that falls outside these limits is subject to additional scrutiny.

In 1988, we chose to sample the accuracy of respiratory care practitioners' charting that they had changed components of the ventilator circuit. The quality assurance coordinator randomly and secretly marked 32 tracheal suction catheters in clinical use. The coordinator returned 24 hours later to see whether the marked catheters had been replaced. These direct observations were compared with the computer charting of tracheal suction catheter changes for the same time periods and the same patients. We found no difference between the direct observations and the computer database, presumptive evidence that the computer database accurately reflects clinical respiratory care practices.

Discussion and Summary

At present, the principal advantage of computer-assisted quality assurance is the acquisition of quality assurance data without resource-consuming chart reviews. A surveillance program like the medical director's alert may reduce morbidity and mortality. Previous research suggests that inadequate oxygen therapy or failures in airway management are important causes of preventable deaths in hospitals.[14] Furthermore, preventable deaths tend to occur among patients who have lower severity-of-illness scores and who are not in ICUs.[15] Thus, surveillance of the entire hospital, as performed by the HIS medical director's alert, may significantly impact hospital mortality related to respiratory care. Future research should critically examine the potential of such computerized systems to favorably change the morbidity and mortality of hospitalized patients.

The departments of respiratory care and medical informatics at LDS Hospital have developed a computer-assisted approach to quality assurance monitoring of respiratory care services. This system provides frequent and consistent samples of a variety of respiratory care data. The immediate needs of patients are addressed through a daily surveillance system (medical director's alert). The departmental quality assurance program utilizes a separate program that monitors clinical indicators of staff performance in terms of stated departmental policies and procedures (rate-based clinical indicators). The availability of an integrated patient database allows these functions to be performed without labor-intensive chart audits.

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Using Belief Networks To Interpret Qualitative Data in the ICU

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Stig K Andersen PhD, and Lawrence M Fagan MD PhD

I. **Introduction**
II. **VentPlan Overview**
III. **Background**
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V. **System Description**
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Introduction

Appropriate therapeutic intervention in the intensive-care unit (ICU) is dependent on accurate interpretation of patient data. If data are not properly collected and documented, treatment decisions are based on misleading information. The effect can have dire consequences for ICU patients receiving treatments such as ventilator therapy for respiratory compromise and intravenous therapy for hemodynamic instability. Because manual methods are generally considered inefficient, many ICUs are installing computer-based systems to improve data collection and documentation.

Standard computer-based systems perform functions such as database management, automated data acquisition, and networked communication across departments. More interesting, from a knowledge-engineering perspective, are systems capable of advanced patient monitoring and therapy planning. Monitoring techniques examine data collected from patients and trigger programs that, for example, detect abnormal trends over time and

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summarize data points into increasing levels of abstraction corresponding to symptoms that warrant intervention.[1,2] Therapy-planning systems organize data into structures used to select the best treatment among possible alternatives or to critique treatment plans under consideration.[3-5]

One challenge in developing clinical decision-making systems is how to represent complex and uncertain relationships inherent in the medical knowledge needed to interpret patient data. Without the proper knowledge representation, the system's ability to interpret data is diminished—producing advice that is of little clinical value. It is difficult to capture all the required knowledge using one formal representation; medical knowledge is too diversified. Well-understood physiologic concepts, such as the influences of heart rate and stroke volume on cardiac output, can be accurately modeled with known mathematical equations. Poorly understood relationships, such as those among many symptoms and interacting diseases, cannot be described by deterministic equations. For these uncertain relationships, probability theory provides a feasible alternative.

We have developed an architecture to combine several knowledge-representation techniques that work in concert to monitor the ICU patient over time and to assist in ventilator management. Implementing this architecture, we have combined techniques of mathematical modeling, probabilistic reasoning, and decision analysis into a prototype system called VentPlan. We use VentPlan as a testbed for investigating the feasibility of this architecture as a model for developing clinical decision-support systems.

In this paper, we describe the construction and testing of the probabilistic reasoning module called VPnet, which is implemented as a belief network. The belief network combines the soundness of probability theory with the flexibility of graph theory into one method for probabilistic inference. The advantages of this combination include an explicit representation of uncertain relationships among domain variables and the ability to represent diverse data types within a single formalism. VPnet uses patient measurements obtained from the bedside to influence calculations performed by VentPlan's mathematical model. VPnet accounts for the uncertainty associated with these measurements and provides a mechanism for representing the meaning of measurements that were acquired previously—a concept we will refer to as the time decay of information. The advantage of VentPlan is that a greater portion of patient data is available for reasoning and advice generation. We describe three areas of VPnet development in this paper—the connection between VentPlan's deterministic model and VPnet, VPnet features of measurement reporting and time decay, and testing of VPnet performance.

VentPlan Overview

VentPlan assists the health-care team in managing patients who are connected to ventilators. Because of the nature of ventilator therapy, the health-care team collects and monitors patient information over time and makes adjustments to treatment in the form of ventilator settings. VentPlan receives patient data as they become available at the bedside, and it recommends ventilator-setting adjustments as changes in physiologic function are detected. VentPlan is also able to predict the effect of recommended adjustments to the ventilator in the form of anticipated steady-state levels of arterial oxygen and carbon dioxide.

An overview of the VentPlan-system architecture is shown in Figure 1.[6] At the core of the system is a mathematical module made up of equations that describe a three-compartment model of the cardiopulmonary circulation of oxygen and carbon dioxide.[7] Inputs to the mathematical module correspond to estimates of physiologic parameters represented directly in the equations. Two components make up the mathematical module. The first, an empirical Bayesian estimator, calculates the best fit for the parameters of its equations using parameter estimates and current laboratory-test values. This fit represents VentPlan's understanding of the patient's current physiologic state. The second component, a simulator, works in conjunction with the plan evaluator to determine the appropriate recommendation for the ventilator settings. The simulator uses the fitted parameters to calculate the physiologic effects of various ventilator settings; the plan evaluator ranks the effects to determine which setting is appropriate. The plan evaluator is a multi-attribute value function that models the trade-offs among various treatments and their associated outcomes.[8] A more complete description of the VentPlan system is available.[9,10]

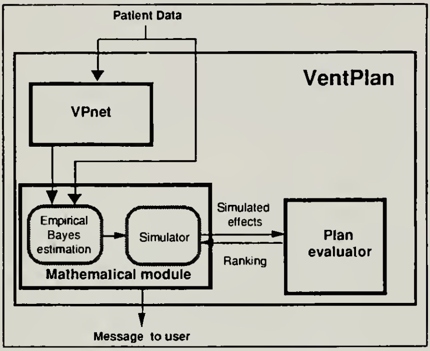

Fig. 1. Overview of VentPlan architecture. Rectangles in bold outline show the relationships of the belief network (VPnet), plan evaluator, and mathematical module. Arrows show the flow of information during a single cycle of data analysis. Adapted from Figure 1 in Reference 6. Reproduced with permission from the copyright holder, American Medical Informatics Association.

The primary purpose of VPnet is to calculate prior parameter estimates needed by the mathematical model. In the absence of VPnet, the mathematical model would have only estimates based on typical ICU patients (population-prior distributions). The network converts inputs, such as the physician's presumptive diagnosis and clinical observations that are not included in the mathematical model, and calculates updated parameter estimates. For example, the diagnosis of pneumonia will lead to an increased estimate of the shunt and an increased chance of sepsis, which, in turn, can change the total peripheral airways resistance and oxygen consumption. The central venous pressure (CVP) is correlated with the left ventricular end-diastolic pressure (LVEDP), which in turn affects the estimate of the cardiac output. The belief-network structure lets us represent these types of relationships, which are hard to include in the mathematical models used to drive the therapy-planning system. In this paper, we address two issues concerning the development of VPnet:

1. design of the belief network to address the needs of the VentPlan system and the ICU domain, and
2. development of tools to assist with performance verification of the belief-network structure and probability distributions.

Background

Belief networks are directed, acyclic graphs in which nodes represent domain variables—such as heart rate and cardiac output—and arcs show important dependencies among these variables.[11] For nodes without predecessors (parents), the probability of each discrete state is given by a prior probability value. For nodes having predecessors, the probability of each state is given by a probability that is conditioned on the values of its immediate predecessors. Conditional probabilities are equivalent to rules of the form "IF (predecessors to a node have a specific set of values), THEN (values of that node are given by a stated probability distribution)." Performing inference in a belief network involves finding the set of probabilities for each node that satisfies the constraints of the assertions and the conditional dependencies. Several algorithms have been described for evaluating belief networks.[11-14]

Using belief networks to develop clinical systems is not without problems. Development is a combined effort involving system developers—both clinical experts and knowledge engineers. Clinical experts define the network structure and assess the conditional dependencies based on their medical experience; knowledge engineers use this information to implement and test the resulting belief network. The number of assessments performed by clinical experts increases exponentially as the number of conditioning parents for each variable increases linearly. Because there is rarely a gold standard for comparison, system developers often use indirect methods to check the accuracy of clinical experts' specifications.

In the following section, we outline stages of belief-network construction that we adapted from evaluation studies of rule-based systems. We use these stages to organize our discussion of prior work done on construction tools that were built to assist knowledge engineers and clinical experts with belief-network development. In the subsequent section, we describe our previous experience with an earlier version of the VentPlan belief network, called DxNet.

A Framework for Belief-Network Construction

Several investigators have outlined general stages for developing rule-based systems.[15,16] We extract from their conclusions a framework for constructing clinical systems using belief networks (Table 1). Associated with each stage is a list of tasks of specific concern when belief networks are used.

Table 1. Stages of Belief-Network Construction for Clinical Systems with Associated Tasks*

Construction Stage	Tasks
1. Top-level design	Selection of domain and target populations Definition of system purpose Specification of input and output variables
2. Development of initial structure	Construction of nodes and arcs Estimation of probabilities Debugging and informal testing of system
3. Structure refinement	Adjustment of probability tables Checks of test-case performance
4. Formal evaluation	Checks of performance on clinical cases Measurement of end-user acceptance

*Table was extracted from conclusions in References 13 and 14.

Stage 1, top-level design, contains three tasks associated with the initial sessions between knowledge engineers and clinical experts—selection of the domain, definition of the purpose of the system, and specification of input and output variables. During this stage the network structure is not defined—the scope and domain of the completed system are discussed in general terms. System performance is judged at an abstract level—for example, what is the clinical relevance of the system output for patients it is intended to assist?

System developers design the initial network structure and perform the preliminary probability assessments during Stage 2 of construction (Table 1). During this stage, clinical experts identify conditional dependencies among the domain variables, and knowledge engineers informally test the system. Several investigators have built tools that assist with developing the initial belief-network structure.[17-21] Each tool has an underlying algorithm for evidence propagation plus the ability to create network structures and probability tables using a graphical interface or text-file specification. Debugging and informal testing features include batch processing of sample cases and graphical displays of resulting distributions. For example, using the HUGIN tool, the user can modify the network structure interactively and can examine the resulting probability distributions in histogram format.[17]

During Stage 3, the system developers refine the initial network structure and begin formal testing of the system performance. Refinement and testing are iterative in nature. Knowledge engineers run test cases through the system, clinical experts examine the output and provide feedback, knowledge engineers modify the network based on the feedback, and the cycle is repeated. SimNet, developed for the Pathfinder system, is one example of a construction tool built to assist developers with structure refinement.[22] SimNet reduces the complexity of structure refinement by partitioning the network into smaller networks that contain only relevant variables. This partitioning confines the refinement and probability-assessment task to one portion of the network—features that discriminate among the presence of similar disease states. By assuming that diseases are mutually exclusive, the SimNet algorithm constructs a combined network that is guaranteed to be consistent with the initial networks.

Once developers have refined the structure of the network, the system specifications are frozen and a formal evaluation begins (Stage 4, Table 1). In general, formal evaluation involves running cases of actual patient data and then comparing system output against a predefined standard. Spiegelhalter and colleagues used assessments performed by three cardiologists to construct a belief network representing congenital heart disease.[23] They then compared the quality of these assessments using data collected prospectively on 200 infants. Based on their analysis, the investigators developed general formulas that use observational data to refine the initial probability assessments.

Prototype Experience: DxNet

The initial belief network used in VentPlan was developed by Beinlich et al using a modified version of the ALARM system—a network that they had developed to detect the occurrence of anesthetic complications during surgical procedures.[24] The adaptation was undertaken because pathologic conditions during anesthesia are similar to those that arise as complications in ventilated ICU patients. The prototype version contained 87 nodes.

During the structure-refinement stage of DxNet construction, we confronted three design limitations that motivated us to develop VPnet.[6] The first was redundancy in knowledge representation between the mathematical module and the belief network. Several physiologic concepts, such as the relationship between oxygen-saturation measurements and arterial-oxygen levels, had a deterministic representation in the mathematical module and a probabilistic representation in the belief network. Although the redundancy was a potential mechanism for verification, the imprecision of the calculated distributions from the network added minimal information when exact mathematical representations were available. In addition, the complexity of the network structure made it difficult to debug.

The second limitation concerned the lack of a temporal representation to discriminate between old and recently acquired data. Recall that VentPlan monitors the patient continuously by tracking data over time. DxNet propagated available evidence through the network with equal weight; there was no notion of time decay for old measurements. A consequence of not representing time decay was that an important measurement obtained infrequently, such as an arterial-blood-gas result from over an hour ago, was quickly overshadowed by a value obtained frequently but with less direct measurement of the physiologic process, such as a recent oxygen-saturation measurement.

The third design limitation resulted from underspecification of ranges for the domain variables. In DxNet, the possible values for each variable were divided into intervals corresponding to ranges, such as low, normal, and high. When reported values crossed into different ranges, the shift in calculated distributions was overemphasized because of the relatively small ratio of ranges compared with the number of possible values. So, for example, when the patient's heart rate rose from 120 to 124 beats per minute, the value shifted from the normal range to the high range. Because only three intervals were specified for heart rate, the shift in distributions was relatively dramatic. This effect propagated through the VentPlan system, resulting in changes in the calculated parameter distributions.

Design Considerations

We developed VPnet to calculate distributions for physiologic parameters used as input to the mathematical module. We describe the correspondence between VPnet nodes and module parameters in the next section. In this section, we discuss two design considerations related to the measurements used as inputs to VPnet:

1. a method for combining uncertain and conflicting evidence, and
2. a method for weighting evidence temporally.

Both issues are related to how ventilator therapy is administered. Once the patient is connected to a ventilator, therapy is a continuous process, ending only when the patient regains normal respiratory function or dies. The health-care team relies on clinical assessment and interpretation of measured data to assess patient response and to guide further therapy. For a computer-based system to assist the team with the tasks of monitoring the patient and recommending ventilator adjustments, the system must be able to capture the dynamic and uncertain characteristics of the measured data. The network must be able to combine qualitative and quantitative measurements while capturing the uncertainty associated with these observations and the uncertainty associated with conclusions drawn from the data. A mechanism is needed for modeling the inherent error rates of monitoring devices and for distinguishing recently reported measurements from older measurements.

System Description

Information stored on ICU patients forms a diversified database. The range of data spans from numeric values obtained from monitoring devices and laboratory tests to Boolean values or character strings representing the presence or absence of complex disease states. Figure 2 summarizes what portion of information from the ICU database is used by the modules inside VentPlan. Inputs to VPnet include data routinely measured by the caregiver at the bedside, such as heart rate and temperature, and information on the presence of disease states, such as hypovolemia and sepsis. Within the mathematical module, measurements of cardiac output and arterial blood gases are passed to the empirical Bayesian estimator, and ventilator settings are passed to the simulator.

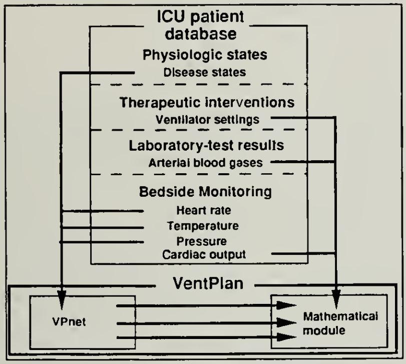

Fig. 2. Flow of information collected in the ICU and passed to VentPlan. The arrows indicate which data types are captured by VPnet and which are captured by the mathematical module.

We now describe in detail the structure and features of VPnet. We start with a description of the basic structure and three types of nodes. We then illustrate how we use the belief-network structure to model the uncertainty in reported data and time decay of previously acquired data. We end with a description of the mechanism for converting VPnet output into parameter estimates that, in turn, serve as inputs to the mathematical module.

Network Structure

The VPnet belief network contains a total of 34 nodes. Figure 3 introduces the basic structure and node types found in VPnet: Diagnostic nodes (black nodes in upper portion of Fig. 3) represent pathologic conditions that occur commonly in ventilated patients. Along the bottom portion are subnetworks shown as flattened gray ovals. Each subnetwork contains time, report, and measured-variable nodes. Time nodes are instantiated with the amount of time that has elapsed since measurements were reported, report nodes are instantiated with values of the latest measurements, and measured-variable nodes are not instantiated—their distributions are calculated from the time and report nodes. We discuss the details of subnetworks in the next two sections. Intermediate nodes are shown as white nodes with black outlines. These nodes combine the evidence from measured-variable nodes with diagnostic information. The distributions of a subset of intermediate nodes (nodes outlined with thick lines in Fig. 3) are used to calculate the parameter estimates that are passed to the mathematical module. VPnet converts the discrete probability distributions for cardiac output, oxygen consumption, and pulmonary shunt into continuous distributions with a mean and variance. The mean and the variance form the parameter estimates that are sent to the mathematical module.

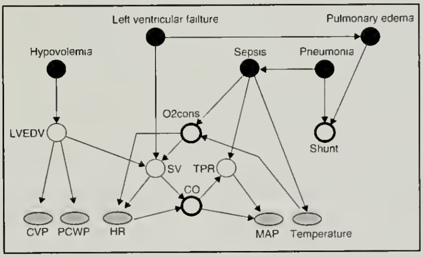

Fig. 3. The VPnet structure. The belief network represents causal relationships with diagnostic nodes (●), intermediate nodes (○ & ○), and subnetworks containing time, report, and measured-variable nodes (⬭). See text for details. CVP: central venous pressure; LVEDV: left-ventricular end-diastolic volume; PCWP: pulmonary capillary wedge pressure; HR: heart rate; SV: stroke volume; CO: cardiac output; TPR: total peripheral resistance; MAP: mean arterial pressure, O_2cons: oxygen consumption. Adapted from Reference 6, Page 786.

Report-Node Structure

We developed the report-node structure to model the distinction between reported measurements and true values. Figure 4 illustrates this structure. Each of the five measured-variable nodes—central venous pressure, pulmonary capillary wedge pressure, heart rate, mean arterial pressure, and temperature—has an associated report node. The report node is instantiated with patient data sent to VPnet. During network propagation, the measured-variable node combines evidence from the report node with additional information from the rest of the network into the updated belief distribution on the true value of the measurement. Measured-variable nodes are divided into three or five categories covering the range of possible physiologic values. Each associated report node covers the same range but is divided into 20 categories.

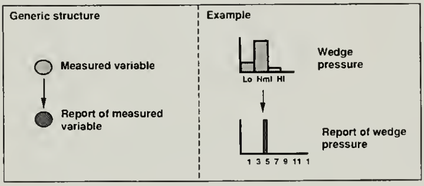

Fig. 4. Association between the report node and the measured-variable node. The left panel shows how the measured-variable node and report node are represented in the belief network. The right panel gives an example of how the ranges are represented for each of the nodes. Lo: low range, Nml: normal range, Hi: high range.

The probability assessment for measured-variable nodes was done by our clinical expert. Probability distributions for report nodes were calculated using a program that we developed in Mathematica, a system for mathematical computation by computer.[25] The program required the probability density function, the variance, and the range and number of categories to calculate report-node distributions. For the results shown in this paper, we used the density function for the normal distribution and the measurement variances listed in Table 2.

We use the report-node structure described in this section for three reasons. First, the inherent error rates of measuring devices are represented explicitly in the variances used to calculate the probability distributions of the report nodes. Second, because a report node covers fewer values per interval compared with the measured-variable node, the transitions in the calculated probability distributions, as reported values cross interval boundaries, were less dramatic than were the corresponding transitions in DxNet. Third, additional evidence propagating from the rest of the network is combined within the actual measured-variable node. This combining feature provides a mechanism for resolution of conflicting evidence.

Table 2. Measurement Variances Used To Calculate VPnet Report-Node Distributions

Report Node	Variance (units)
Central venous pressure	± 4 mm Hg
Pulmonary capillary wedge pressure	± 1 mm Hg
Heart rate	± 25 beats/minute
Mean arterial pressure	± 25 mm Hg
Temperature	± 0.09 °C

Time-Node Structure

VentPlan is a system that tracks patient data over time and recommends modifications to ventilator settings as changes in physiologic function are detected. Because the system tracks data over time, any call to VPnet includes measurements from differing time intervals. A typical evidence set consists of measurements recently reported, such as heart rate and blood pressure, plus measurements from past reports, such as temperature or pulmonary wedge pressure. The former are collected frequently (many reports per hour), whereas the latter are recorded less frequently (once every few hours). Each measurement offers some evidence of the patient's current physiologic state, but each has a differing degree of import.

The method we use for incorporating time decay of past measurements is illustrated in Figure 5. Each report node has an associated time-decay node. Each time-decay node has binary values—it is false when the measurement is first reported, it is true when the measurement was reported prior to the current time. As time passes, the probability increases that reported measurements no longer represent the current true value of the measured variable. The passage of time is simulated by shifting the bulk of the distribution gradually toward a true value for the time-decay node. As this shift occurs, the measured-variable node becomes conditionally independent of the report node. A time-lapse node determines how the probabilities are distributed between the values of the time-decay node. The values for the time-lapse node correspond to increasing half-lives of exponential decay. The time constants vary for each report node. For example, heart rate and blood pressure have time constants of 10 minutes, whereas pulmonary pressure and temperature have time constants of 1 hour.

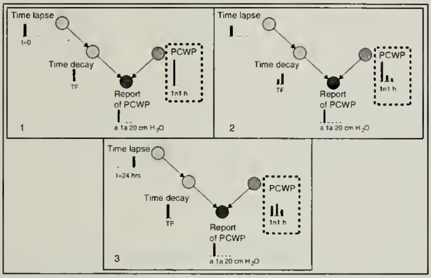

Fig. 5. The structure of the VPnet time-decay and time-lapse nodes. In this example, the measured-variable node is the PCWP, and the report of PCWP is set to 4 cm H_2O. The time-lapse node for PCWP is instantiated with time lapse = current (Panel 1), time lapse = one half-life at t = 10 minutes (Panel 2), and time lapse = greater than three half-lives at t = 24 hours (bottom panel). The distributions on the PCWP are boxed within a dashed rectangle in each panel to highlight the reduced influence of the report of PCWP on the PCWP node as the report of PCWP ages. Adapted from Reference 6, Page 787.

Connection between VPnet and Mathematical Module

Once evidence has propagated through the network to establish values for the nodes corresponding to parameters of the mathematical model, these in turn must be converted to continuous distributions. The steps of this process are illustrated in Figure 6. There is a one-to-one correspondence between the subset of VPnet intermediate nodes and three module parameters—cardiac output, oxygen consumption, and pulmonary shunt. For each corresponding node, VPnet calculates a probability distribution (P_1, ..., P_n, where n equals the number of categories) over the range of possible discrete values (a_1..., a_n+1). We convert the discrete distributions into normal distributions by calculating the mean and the variance.

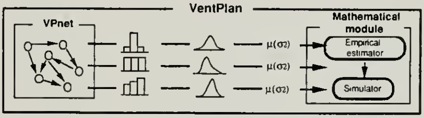

Fig. 6. Connection between the mathematical module and VPnet. Broken arrows indicate the steps that are performed in converting discrete probability distributions into continuous normal distributions, which are used by the mathematical module.

The means and the variances form the set of parameter estimates that are passed to the mathematical module. The mathematical module combines the estimates with arterial-blood-gas data and cardiac-output measurements to calculate the physiologic-model parameters. Table 3 shows the parameter estimates that VPnet calculated using the method described in this section for 10 patient scenarios.[6]

Status Report

VPnet is implemented in LightSpeed Pascal on the Apple Macintosh II, using the Lauritzen-Spiegelhalter algorithm for network evaluation.[14] The current implementation evaluates the network with any possible evidence set in less than 2 seconds. The next section describes the tools that we used to construct and test the network. The subsequent section describes the tools that we used to construct a library of patient cases that will be used to evaluate the effect of VPnet on VentPlan performance.

VPnet Network-Structure Construction and Testing

Construction of the network structure and initial probability assessment were performed primarily using HUGIN and one clinical expert.[17] The HUGIN environment has a graphical interface that allows generation of test cases with immediate network update and display of node distributions in histo-

Table 3. The Results of Evaluating the Network with Measured-Variable Nodes and Diagnosis Nodes Directly Instantiated, Showing the Effect of Evidence on Parameter Distributions*

Evidence						Parameter Mean		
HR	MAP	Temp	CVP	PCWP	DX	Cardiac Output (L/min)	Shunt Fraction	Oxygen Consumption (mL/min)
–	–	–	–	–	–	5.3 (2.2)	0.07 (0.07)	281 (83)
N	N	N	–	–	–	5.2 (1.6)	0.07 (0.07)	248 (53)
H	N	H	–	–	–	6.8 (2.2)	0.07 (0.08)	383 (74)
H	N	H	–	L	–	6.4 (2.3)	0.07 (0.08)	379 (76)
H	N	H	–	L	Sepsis	7.4 (1.9)	0.16 (0.11)	383 (74)
H	L	VH	–	L	Sepsis	4.9 (2.4)	0.16 (0.12)	388 (71)
H	N	N	–	–	–	5.6 (2.3)	0.07 (0.07)	291 (84)
H	N	N	H	–	–	5.5 (2.3)	0.07 (0.07)	291 (84)
VH	L	N	H	H	–	3.9 (2.3)	0.07 (0.07)	278 (72)
VH	N	N	H	H	Pul Edema	3.5 (1.9)	0.16 (0.11)	276 (69)

*Numbers are mean (SD). VH: very high measurement. H: high measurement. N: normal measurement. L: low measurement. –: no value asserted. HR: heart rate. MAP: mean arterial pressure. Temp: temperature. CVP: central venous pressure. PCWP: pulmonary capillary wedge pressure. DX: diagnosis. Pul Edema: diagnosis of pulmonary edema. Adapted from Reference 24, Page 788, and reproduced with permission from American Medical Informatics Association.

gram format. The dynamic nature of the interaction enabled our clinical expert to verify the performance of the initial network structure by instantiating various nodes and observing network behavior. Sample case scenarios, similar to those shown in Table 3, were created to simulate progressive worsening of disease states modeled in the network. So, for example, in the presence of normal values for heart rate, blood pressure, and temperature, the network calculated intermediate-node probabilities with means in the normal range, and diagnostic node-distributions with means shifted toward absence of disease. This informal testing allowed us to verify the dependencies modeled among the variables represented in VPnet's network structure to the satisfaction of our domain expert.

Once the internal structure of the network was tested, we developed two versions of a questionnaire, each containing a separate subset of eight patient cases. Each patient case included a single diagnosis and a set of hemodynamic parameters. Physicians were asked to estimate the distribution of three physiologic variables for each patient case. The variables corresponded to VPnet nodes used to calculate parameter estimates passed to the mathematical module. By asking for these distributions, we were able to compare the mean and variance calculated by VPnet with the midpoint and range specified by physicians. Questionnaires were distributed to five internists in the Division of Health Services Research and to one ICU intensivist in our institution. Figure 7 details the relationship between responses and calculated means for each of the 16 cases. Each point on the graphs plots the correspondence between the calculated mean and the midpoint average for all physicians responding to that case. The correlation coefficients for the three parameters fell between 0.67 and 0.81. VPnet routinely calculated a slightly lower cardiac output and a slightly higher shunt than was estimated by the physicians. Differences in oxygen-consumption distributions were consistently small, with a tighter range specified by the calculated VPnet variance than by the physicians.

Patient-Case Library Construction

We installed an automated data-collection system in the surgical ICU at the Veteran's Administration Hospital in Palo Alto, California. The

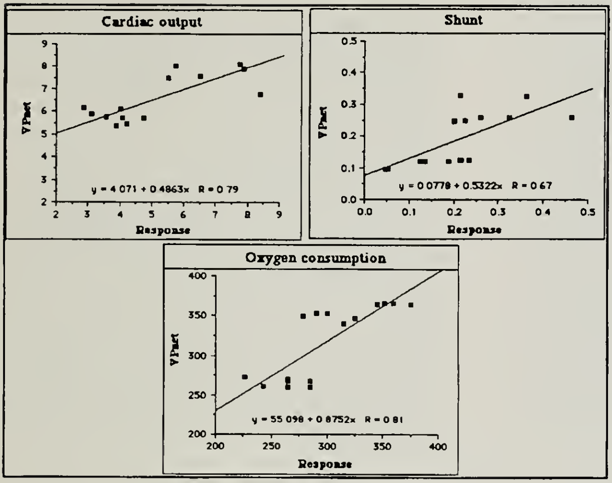

Fig. 7. Individual data points for all parameters. Each panel corresponds to a different physiologic parameter, labeled at the top. The graphs plot the average for the questionnaire responses (x-axis) versus the mean calculated by VPnet (16 cases, six respondents). The linear equation and correlation coefficient are also given for each parameter.

system includes the necessary hardware and software to interface directly with the bedside monitor, ventilator, and pulse oximeter. We can scan each of these devices as frequently as every 5 seconds—and can store the time, value, and description of each measurement collected. Our longest case runs approximately 16 hours, with data collection ranging from once every 10 seconds to once per minute. We collect blood-pressure measurements every 10 seconds to correlate the arterial-blood-gas reports with time of the actual blood draw. Blood-gas information is collected manually and must be integrated into each test case based on when the specimen was collected, not on when the results become available.

We currently have in our patient library 14 cases that we collected using this system. The library provided us with a rich source of data for testing VentPlan performance during the early stages of development. We are using the patient library to compare VentPlan performance with and without the belief network. Because parameter estimates have the same form no matter how they are generated, it is possible to run VentPlan by replacing distributions calculated from VPnet with distributions that reflect values typically found in ventilated ICU patients.

Lessons Learned

We have described a belief network designed to model the relationships among direct measurements of physiologic variables and concepts of intermediate physiologic states and disease processes. Based on the testing completed to date, we identify three major areas needing further development. The first is the representation of the complexity of disease states. VPnet would benefit from a richer representation of disease states that included severity of disease; it would also benefit from the addition of more of the disease states that commonly occur in ICU patients.

The second limitation stems from assumptions we make concerning a normal distribution in the parameters we calculate. Many variables cannot be negative and thus have skewed distributions not modeled accurately by a normal curve—for example, the shunt varies from 0 to 1, but the normal physiologic value is near 0. We are testing solutions to this problem with the use of data transformations.

The third limitation is the need for computer-based tools for formal evaluation of VPnet performance. An important consideration of formal evaluation is that domain experts are likely to be unfamiliar with probabilistic reasoning and with interpretation of probability distributions. We need a method that hides the network structure and cryptic output and draws attention only to information pertinent to the evaluation task. A computer-based tool would provide two main advantages over the questionnaire. First, the case-selection task would be left to the clinical expert. An input screen would allow the expert to create a set of VPnet inputs dynamically. Inputs include values from bedside measurements, such as heart rate and blood pressure, as well as diagnostic information. Second, by displaying the network output to the clinician and allowing the opportunity for modification, we can capture, store, and retrieve for later reuse, a log of each session. This feature would enable us to make maximal and efficient use of clinical-expert feedback.

Conclusions

VentPlan allows us to test the feasibility of a combined approach for reasoning techniques in building therapy-planning systems for ventilator management. We use the mathematical module to reason about the physiology of oxygen transport, the plan evaluator to account for trade-offs associated with ventilator therapy, and the belief network to make use of patient-specific measurements and to capture the uncertain influences among physiologic concepts.

We have described a method for using belief networks to modify parameter estimates passed to the mathematical module using patient-specific evidence. We have described methods that model the uncertainty of reported evidence and the time decay of past information. Using a sample of physician estimates, we compared the calculated VPnet distributions and found encouraging preliminary results. We now face the task of a formal evaluation of VPnet as a belief network and as a functioning part of VentPlan.

Formal evaluation has been one of the biggest challenges for system developers. For belief networks, evaluation primarily means verifying prior and conditional probability distributions; for therapy-planning systems such as VentPlan, it means developing measurements that test the accuracy of complex systems. Because the luxury of a gold-standard comparison often is not available, we look to approximate methods. The use of estimates obtained from clinical experts is one approach. Our experience and that of other investigators indicate that, when collected with the proper tools, correlation with human judgment can contribute to verification of system performance.

We have tested the performance of VentPlan's mathematical module and plan evaluator under conditions of fixed prior-parameter distributions.[6] We have not yet verified the effect of VPnet on VentPlan performance. The challenge lies in creating test-case scenarios that simulate real-life data-collection activities of the ICU environment and in developing standards for measuring performance. In exploring these issues, we hope to gain insight into important design requirements for general evaluation tools.

ACKNOWLEDGMENTS

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Microprocessor-Based Mechanical Ventilation

Warren G Sanborn PhD

I. History and Development of Ventilator Function
  A. Vesalius through the 1970s
  B. 1980—The Beginning of the Third Generation
II. The Pneumatic Subsystem
  A. The Mechanics of Flow Delivery
  B. Feedback Control
  C. Enhanced Control through Learning
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History and Development of Ventilator Function

Vesalius through the 1970s

Before we attempt to determine why the function of a ventilator might be enhanced or improved by the inclusion of one or more microprocessors in the design of the device, it may prove worthwhile to ask what ventilators do. Perhaps they do more today than they did in the past.

The history of ventilation is well known and needs only to be referenced here (see also the thorough bibliography by Kacmarek[1]). In the middle of the 16th century, the foundations of modern medical thinking were cast by Andreas Vesalius.[2] His two tomes are inspirational, even today. He presented the first documented evidence that air blown into the trachea of a dog could maintain the steady and vigorous rhythm of its heart. As with so many important initial discoveries, an immediate full and complete understanding of the original observations was lacking.

Vesalius's observations were rediscovered and elaborated on by Hook in the middle of the 17th century.[3] As an interesting aside, it appears that Hook serendipitously discovered high-frequency

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ventilation. But like Vesalius some 100-plus years earlier, he could only describe, rather than explain, his observations.

Just prior to the start of the 20th century, commercial ventilation became available with the introduction of the Fell O'Dwyer Pulmoflator.[4(184-190)] Interestingly, the commercialization of ventilation was no longer limited by functional misunderstanding; rather, the product was delayed due to the lack of a means to introduce breathing gas distal to the larynx. During the first half of the 1900s, ventilators became motorized, but they did little more than force gas into the lungs and allow it to escape passively.

The polio epidemics of the 1950s, both in the United States and in Europe, provided the impetus to legitimize positive-pressure ventilation in the eyes of the medical community.[4(206-220)] Soon, a variety of ventilators became available, some pressure-based, some volume-based, and some with patient-triggering. Even the introduction of feedback control of gas delivery did little to enable the practitioner to manage the patient's work of breathing. Ironically, the work of breathing spontaneously was best managed by providing the patient with continuous flow, which could be achieved without a mechanized ventilator.[5] This scenario persisted up to approximately 1980. Most certainly, the period 1950-1980 saw many noteworthy improvements in ventilator design.

Monitoring began with just an airway-pressure manometer. Later, alarming for low and high pressure was added. Exhaled-tidal-volume monitoring was added and extended to include alarming for low exhaled tidal volume (V_T) and minute ventilation. A fail-to-cycle alarm provided a measure of added security. The addition of metabolic measurements in the late 1970s marked a departure from the ventilator as a single-function device. However, the relatively low acceptance of this feature, even to this day, represents concern on the part of the practitioner that the ventilator as a metabolic monitor lacks the necessary accuracy. Notwithstanding the improvements in design, monitoring, and alarming, the ventilator strayed little from a single-function product. The focus of the ventilator, despite repeated attempts at enhanced function[6] and design,[4(614-618)] remained that of gas delivery.

1980—The Beginning of the Third Generation

The decade that began with 1980 saw more than the application of advanced engineering to ventilator design. I have discussed the broad and general aspects of this theme elsewhere[7] and will refrain from detailed discussion in this paper. In general terms, several factors converged as the 1980s began. Clinicians and engineers had been acutely aware of the potential of applying digital, microprocessor-based electronics to ventilator design, but they were inhibited from acting too hastily, as price and computing power remained cost-ineffective until approximately 1980. Once this barrier was breached, the technical limitations that had thwarted the design of a ventilator around one or more microprocessors vanished. A second theme quickly crystallized: The ventilator could be visualized—albeit only hazily—as something more than just a gas-delivery machine. And lastly, there were notable advances in the design of gas-delivery valves (flow-control valves) that were particularly advantageous to the implementation of more responsive gas-delivery systems.

For the first time, a ventilator could be visualized as something more than a single-function device, a box that delivered breathing gas to an intubated patient. (Even this last notion, intubation, is changing; with greater frequency, the literature speaks about successful face-mask ventilation.[8]) Because of the unique role of the ventilator in the life-support, ICU setting, it becomes more and more acceptable to think of the ventilator as a system whose goal is the safe and effective management of the patient's respiratory function. This line of thinking implies that the ventilator has become a tool for the management of the work of breathing and gas exchange. Given the anticipated fruition of many of today's research projects, this expectation broadens yet further. It seems quite reasonable to expect that the ventilator of the future will report the patient's metabolic state; manage oxygen delivery; calculate cardiac output; synchronize breath delivery with the cardiac cycle to maximize cardiac output, or at least minimize the potentially deleterious impact of positive-pressure ventilation on cardiac hemodynamics; and, lastly, perform all of these functions automatically or at least presenting consensus-based advisory messages to the practitioner for confirmation or modification. (Recent and apparently positive results with a 'simple,' protocol-based ventilator-management program[9,10] suggest that this approach may have far-reaching implications regarding standards of care.)

At this point, it is fair to ask what microprocessor-based electronics and the aforementioned ideas have in common. The answer, I would suggest, is literally everything. The operation and control of the system just enumerated are hardly imaginable without the diverse power and flexibility—the intelligence—of a microprocessor-based (or multiprocessor-based) design. Individual subsystems within the overall scheme could be analog- or digital-based, but very likely a single- or multi-processor design would be employed to run the system. In the remainder of this paper, I will discuss ventilator design, current and future, subsystem by subsystem, with respect to the advantages of microprocessor-based control.

The Pneumatic Subsystem

The Mechanics of Flow Delivery

Whatever else a ventilator may be, at its very heart it is a gas-delivery system. If the aim of a ventilator is the management of the work of breathing and gas exchange (here I am referring to an ICU-level device), there is little room for trading degraded performance for price reduction. Outside of the ICU, this trade-off may make economic sense, but the cost-performance ratio is now declining so rapidly that a design offering mediocre performance costs only fractionally less than one offering high performance. Even in home care, where patients presumably can more readily be integrated with lower-performance systems, more efficient designs (currently in prototype) appear to offer superior performance for the same price. (See Reference 11 for recent studies of assist-control ventilation and the work of breathing.)

As recently as a few years ago, the term "pneumatic subsystem" uniquely defined the means by which gas delivery to the patient was controlled. Today, it is more correct to include the expiratory valve as another element enhancing the gas-delivery function. I will first discuss gas delivery and exhalation as separate functions—and then combine them later.

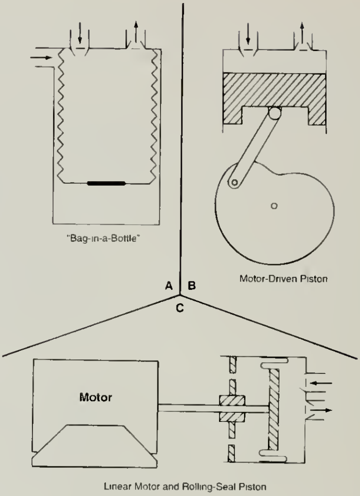

Fig. 1. Three types of volume-displacement, pneumatic designs. Panel A: the "bag-in-a-chamber" implementation. As the bag expands, it fills with mixed gas. Pressurization of the chamber outside of the bag causes it to collapse, driving gas into the patient's lungs. Panel B: the piston-and-cylinder implementation. As the piston moves to the rear, mixed gas enters the cylinder. Forward movement of the piston expels the gas, driving it into the patient's lungs. Panel C: the linear-motor, rolling-seal implementation. The operations of this and the piston-and-cylinder designs are similar, except for the virtual elimination of piston-to-cylinder friction due to the rolling seal. Driven by the linear motor, the system's responsiveness rivals that of proportional-valve designs (see Fig. 2).

Throughout the evolution of pneumatic subsystems, several common designs stand out. From an engineering perspective, it is convenient to divide these designs into two broad categories: volume-controllers and flow-controllers. Figure 1 illustrates three examples of volume-controllers. Panel A represents volume displacement via a collapsible bag. The bellows or bag typically contains the breathing gas, which is expelled by pressurizing the outer chamber. Because the response time and flexibility of flow-control valves are generally superior to the "bag-in-a-chamber," this once-popular design is rarely found in ICU-type ventilators today. Typical examples of the bag-in-a-chamber include the MA-1 and 2 and the Monaghan 225 ventilators. Most anesthesia ventilators employ this design because the bag effectively separates the breathing gas from any mechanical contamination.

Interestingly, the motor-driven piston (Panel B) has possibilities not realized earlier because of its dependence on piston-to-cylinder friction and a relatively slowly responding motor and crankshaft assembly. This design is still found in the Emerson ventilators and some home-care ventilators. Lead-screw motors have improved the flexibility and response time, but rotational inertia still places limitations on the design. Whatever the improvements in motor configuration, it seems probable that the piston-in-a-cylinder design will lack the features required for an ICU-type ventilator.

The Hillman piston-and-motor design[4(614-618)] opened new opportunities by replacing the conventional, rotating-armature motor with a linear motor. By combining a linear motor with a rolling-seal piston[12] (Panel C), the positive-displacement pneumatic subsystem assumes even greater flexibility and responsiveness. Recently published, preliminary information speaks well of this design concept.[13-15]

Most present-day ICU-type ventilators are based on flow-control valves, illustrated in Panels A, B, and C of Figure 2. These valves proportionally open and close, driven by a variety of motor-based mechanisms. For example, the valves in the Puritan-Bennett 7200, the Hamilton Veolar and Amadeus, the Dräger EVITA and IRISA, and the Siemens SV300 incorporate a flow-metering orifice that is directly actuated by an armature moving linearly in a magnetic field (Panel A). The flow-metering orifice in the Infrasonics Adult Star is directly coupled to a stepper motor. In the Siemens 900 series, flow metering is accomplished by a stepper-motor-driven scissor valve that pinches a silicon tube. In the Bear 5 and 1000 and the Bird 6400 and 8400, flow control is accomplished by valves that are actuated by a stepper-motor-driven cam (Panel B).

A third configuration, illustrated by Panel C in Figure 2, utilizes digital valves. In this design, each valve is either open or closed. When open, a given valve produces only one 'calibrated' flow. Thus, if a three-valve design were selected, a total of eight unique flow values could be produced, including zero. For example, if a specification called for 7 L/min maximum, with a resolution of 1 L/min, one valve would be calibrated to deliver 1 L/min, the second valve 2 L/min, and the third 4 L/min (yielding 0, 1, 2, 3, 4, 5, 6, 7 L/min with the appropriate sequencing of the valves). Although this design appears charmingly simple, two primary issues must be solved: accuracy and component reliability. Maintenance of the supply pressure in excess of the critical pressure for the largest orifice solves the first issue. The second issue, that of the increase of the number of critical components directly controlling flow from one or two to five valves in the

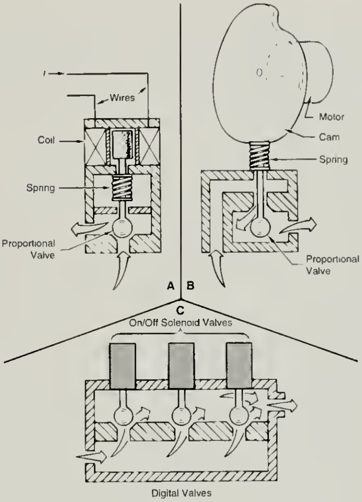

Fig. 2. Three types of pneumatic valves used to control flow. Panel A: one type of proportional solenoid valve. In this design, a controllable electrical current flows through the coil, resulting in a magnetic field. The strength of the magnetic field causes the armature to assume a specified position. With the armature and valve poppet physically connected, this assembly is the only moving part. Coil and armature designs vary, as do the strategies used to fix the position of the poppet. Panel B: an externally actuated proportional valve. Several different motor and cam schemes have been designed with which to control the position of the poppet. The stepper motor and scissor design, implemented in the Siemens 900 series ventilators, represents an early, highly successful strategy. Panel C: the digital-valve, on-or-off concept. With each valve controlling a critical orifice and hence specified flow, the number of discrete flow steps (including zero) becomes 2^n (where n = number of valves). A 9-valve design, with 0.5-L/min flow increments between valves, yields a flow range of 0 to 255 L/min and a resolution of 0.5 L/min.

case of the Infrasonics Infant Star, is solved if the n valves together exhibit reliability equal to or greater than that of one or two sophisticated valves. The digital-valve design is attractive because each valve can be considerably less sophisticated than a 'full-range' proportional valve.

Once the configuration of the pneumatic subsystem has been specified, the next issue is that of the control strategy and the means of control. Given the complexity of today's ICU-type ventilators (and home-care ventilators, for that matter), it should be no surprise that the gas-delivery system is under the control of one or more microprocessors. The question confronting the designer is how to control the flow-metering valve (or valves) or the motor-piston assembly. Prior to 1980, virtually all designs were based on analog circuitry. Once high-powered microprocessors became cost-effective in the medical setting, designers could choose either analog control or digital control. For example, the flow-control valves in Puritan-Bennett's 7200 series ventilators are run by a microprocessor (ie, digital electronics), whereas the flow-control valve in the Hamilton Veolar and Amadeus is run by analog electronics. From the patient's point of view, such issues are largely academic. Central to the patient is the ability of a given ventilator to provide exquisite management of his or her work of breathing. In this regard, both digital control and analog control have the capacity to provide the desired level of performance.

That last statement begs further elaboration. When microprocessors are chosen as design elements, it is not because of their mysterious qualities. Long before microprocessors became fashionable, highly reliable and precise analog circuits were performing highly reliable and precise feats of electronic switching and control. The mystery attributed to the microprocessor stems from its extraordinary versatility. Apple and Microsoft's windowed environments, Lotus 1-2-3, word processing, computer-aided design (CAD), computer-aided engineering (CAE), computer-aided manufacturing (CAM), video games, and the like are results of the microprocessor's ability to execute digitally encoded instructions. To perform their wonderful 'magic,' microprocessors run under the direction of sophisticated software programs in conjunction with voluminous memory. If you wish to be awakened in the morning with a musical tone, followed by a voice that says, "The time is 7:30 AM," to have the coffee started at 7:55, the TV turned on at 8:00, and a taxi phoned for at 8:30, a microprocessor-based design is the answer. What appears like magic on the macrolevel is sophisticated engineering on the microlevel.

What the microprocessor means to a ventilator is versatility of control and function. (See Reference 16 for a statement about the essential features of a ventilator's gas delivery system.) Microprocessor control does not necessarily guarantee outstanding performance from a ventilator's pneumatic subsystem. Once the pneumatic subsystem receives the signal to cycle on and deliver a breath, gas delivery is a function of both the electronic control strategy and valve performance. Figure 3 shows the inspiratory phases of pressure-supported breaths supplied by four third-generation ventilators. Puritan-Bennett's computer-controlled breathing lung, nicknamed Sybl, was set up to request a spontaneous breath having an inspiratory time of approximately 1.0 second and a tidal volume of approximately 300 mL (measured without an ET tube). Once Sybl and a given ventilator were in a steady state, the ventilator's level of pressure support was adjusted to yield a tidal volume of 0.8 L. (This adjustment was made to correct for the difference in ventilator algorithms that dictate the final tidal volume in the face of a sustained inspiratory effort. The adjustments were minor, in any case, and did not cause any significant changes in the trigger work.)

The tracings in Figure 3 illustrate significant differences in the early performance of a ventilator's ability to deliver gas to Sybl. (The location of the airway-pressure sensor can be ignored [see References 17 and 18 for information on the possible effects of sensing location on the work of breathing] because humidifiers were deliberately omitted from the configuration of the patient circuits.) Two factors account for delay between the demand for flow and the delivery of flow to Sybl: the physical design of each unique pneumatic subsystem (metering valve, actuating motor, and pressure at the head of the valve) and the flow-control algorithm. The tracings of Figure 3 result from both factors operating together.

To uncouple the two factors, each ventilator was triggered on to deliver gas to the room (ie, the pa-

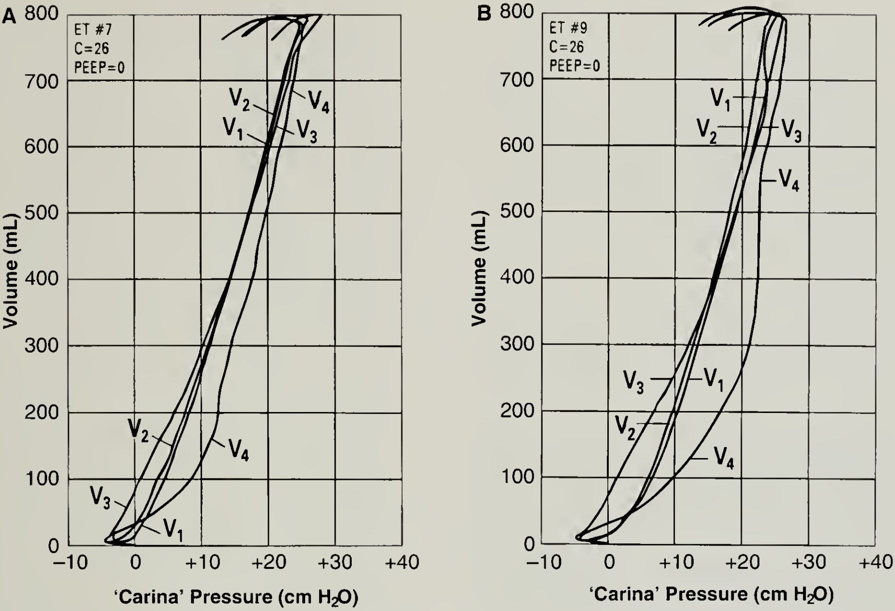

Fig. 3. The inspiratory phases of pressure-supported breaths as revealed by pressure-volume loops. Spontaneous breaths were simulated by Puritan-Bennett's computer-controlled breathing lung (nicknamed Sybl). Breathing free of a ventilator and an endotracheal tube, Sybl was programmed to generate a V_T of approximately 300 mL. Sybl received no further adjustment during the study. Sybl's inspiratory effort generated a high demand for flow early in the breath (non-square but steep rise) then tapered down to near zero at end-inspiration (a ⟍_-like shape). Each ventilator supported Sybl's breathing via #7 and #9 ET tubes. Pressure was measured at the simulated carina position, and pressure sensitivity was set to 1 cm H_2O if adjustable. Once breathing began, pressure-support ventilation was initiated and titrated to the level that boosted Sybl's tidal volume to 800 mL. With the #7 ET tube, the pressures ranged from 26 to 29 cm H_2O, and with the #9 ET tube, 24 to 28 cm H_2O. This approach to PSV was thought to account for minor differences in the design of each ventilator's breath-delivery algorithm. The shapes of the P-V traces early in the breaths suggest that the protocol for the data shown in Figure 4 may have achieved open-loop control, at least in the early milliseconds after the valves opened. Note that the trigger work observed in this figure parallels the order of early flow in Figure 4. For early flow, V1 > V4 > V2 >> V3, and for early trigger work (#7 ET Tube), V1 < V2 < V4 << V3, (#9 ET tube) V1 ~ V2 < V4 << V3.

tient Y was open to the room). The tracings resulting from this protocol appear in Figure 4. Each tracing was aligned with the t_0 reference line. The earliest detectable flow appeared at approximately t_0 + 40 ms; and by t_0 + 45 ms flows for the most responsive ventilators reached 20 L/min. Referring to the 80-L/min flow level, one sees that the order of flow responsiveness is V1 > V4 > V2 >> V3. The corresponding intervals (t_0 = reference) measure 55, 65, 86, and 171 ms, respectively. Figures 3 and 4 taken together suggest that the trigger and early work seen in the pressure-supported breaths bear some relationship to the constraints placed on the early-flow capability by each ventilator's pneumatic subsystem. V1 exhibits a noticeable edge over V2 and V4 with the #7 ET tube. With the #9 ET tube, V1's edge is still noticeable, but less so. V2 now demonstrates a substantial advantage over V4. With either ET tube, V3 operates at a fundamental disadvantage that cannot be masked by algorithmic design. (Note: these data do not imply that V1 will demonstrate an edge in triggering work under all conditions. This specific protocol was selected because it stressed each ventilator's ability to open its

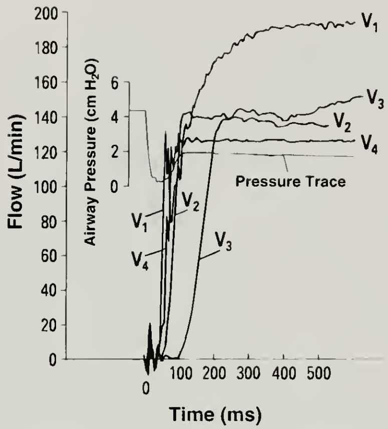

Fig. 4. Quasi-open-loop flow from the pneumatic systems of four third-generation ventilators. Flow was measured by a Fleisch #3 pneumotachograph fitted to the "machine outlet" of each ventilator. The patient circuit consisted of 30-inch (76-cm) inspiratory and expiratory tubes. Airway pressure was recorded from a port located immediately distal to the pneumotachometer. When possible, the ventilators' airway sensing tubes were connected to one of several positions (proximal to the pneumotachometer, distal to the pneumotachometer, at the patient Y, and at the distal end of the exhalation tube) while the flow-time traces were recorded. In some cases the later stages of flow were impacted by the location of the sensing port, but the rapid rise of flow to 120 L/min exhibited no change. To maximize the error signal responsible for opening the flow-control valve(s), pressure support was set to 70 cm H_2O. To initiate a spontaneous breath, PEEP was set to a nominal 5 cm H_2O. Two methods were used to trigger the ventilator on: the removal of my thumb from the common port of the patient Y, and the opening of a piloted valve attached to the Y. Both methods yielded identical tracings. Each ventilator was triggered on at least 5 times. Minor variations in the later stages of flow were observed, but the early traces always exhibited the relationships shown in the figure. If any of the ventilators had a provision for the control of the pressure-rise time, it was set to the maximum. Although true open-loop flow could not be verified, the constancy of early flow suggested that early flow achieved its maximal values.

flow-control valves in response to a high demand for flow. At lower levels of pressure support, and particularly with smaller-diameter ET tubes, the performance of V1, V2, and V4 could alter substantially. The apparent deficit in V3's early performance could prove difficult to correct because its slow-to-open response may be inherent in the design of its flow-control valving, a factor that algorithmic design cannot overcome.) If the flows required to sustain the pressure-supported breath are limited only by rates of change of flow and not by early-flow limitations, the post-trigger phase of the inspiratory P-V traces do primarily reflect each ventilator's algorithmic-control strategies. Figure 3 demonstrates, for example, that V4 manages to sustain a more aggressive flow delivery once the triggering event has been initiated. During the latter half of the protocol breath, V1, V2, and V3 exhibited roughly equivalent performance.

Once gas delivery begins, the control of pressure or the flow into the patient Y is largely a function of the feedback loop from the patient circuit to the gas-delivery valve(s). (Although airway pressure, P_{aw}, is the parameter most commonly fed back to control flow—and hence the monitored pressure—an algorithm has been developed recently in which flow is fed back to control flow.[19])

From the engineering standpoint, breath delivery requires conscious trade-offs. If flow is too aggressively delivered, both flow and pressure will oscillate. Minor oscillation causes few problems, because the artificial airway represents a significant impedance that consequently degrades the oscillations to a level that may be insignificant at the carina. Severe oscillation, however, leads to perceptible sensation in the airway. A design that achieves optimal flow and pressure with a given artificial airway, patient compliance, and airways resistance will underdeliver flow if the total impedance (the result of combining these two resistances with the lung-thorax compliance) decreases, and it will overdeliver flow if the combined impedance increases. No currently available ventilator optimally matches delivered flow with the patient's demand for flow over the entire range of input impedances, although the Dräger IRISA, Siemens SV300, and the soon-to-be-released Bear 1000 do offer a pressure-rise time adjustment.[20, 21]

As research deepens on the topic of the work of breathing, the ability to match patient and ventilator for optimal management appears to be gaining support, at least in some research-oriented circles (see Reference 22 for the abstract reporting one study about the use of adjustable pressure-rise time, and Reference 23 for the abstract reporting on slight adjustments in pressure support that yield similar results). Whether adjustable pressure-rise time becomes a clinician-set variable will depend on practitioner demand and reports showing efficacy. Were this feature to become 'essential,' two obvious strategies would be available to the designer. One could be called the 'stick-shift' approach and the other the 'automatic-shift' approach. With the stick-shift approach, the clinician would observe pressure and flow waveforms on a video screen. If pressure rose too slowly, the clinician could choose a faster pressure-rise time. In this scenario, the appropriate selection of rise time would be achieved through trial and error. With practice, the adjustment process would become quite routine. (The reader should be aware that the maximal rise of pressure is not necessarily a desirable goal [also see References 20 and 21]. My personal experience with high levels of pressure support suggests that a too-rapid rise in pressure produces a sensation of impending overinflation.) With the automatic-shift approach, the practitioner would select, for example, a gentle, or moderate, or moderately aggressive, or aggressive rise time; and the ventilator would analyze its performance, iterating the internal setting for rise time to the appropriate value as dictated by the practitioner's selection. By the use of a running average of some number of successive, previous breaths, pressure-rise time could be maintained well within acceptable limits.

The interested reader may ask how a microprocessor fits into a design for pressure-rise time. Although an exact answer lies beyond the scope of this article, an operational understanding is appropriate. For the stick-shift approach, the settings and the exact behavior of airway pressure versus time would be studied and quantified over a range—from impedances appropriate for small-diameter artificial airways, high airways resistance, and low lung-thorax compliance to impedances appropriate for large-diameter artificial airways, low airways resistance, and high lung-thorax compliance. A range of flow-gain settings would be selected that allowed the complex control algorithms to span the desired spectrum of performance. Perhaps nine such discrete algorithms would be defined. Each one would be assigned a number, say from 1 to 9, to be chosen from a menu or other 'protected' selector device. A selection would be read by the microprocessor, and software would set the appropriate gain setting into the ventilator's currently active parameters. To provide additional safety, a power-on, new-patient message would be displayed to the practitioner when the power switch was set to ON. An internal, software-based (read: microprocessor-executed), logic-choice matrix would ensure that a safe value for flow gain was always set for a new patient. Only after ventilation began would software allow changes to the rise-time algorithm. Yet this same logic-choice matrix would permit a non-default selection when a change was properly addressed. In practice, the clinician would observe the current pressure and flow waveforms, assess whether the patient might benefit from an adjustment of rise time, and make the appropriate choice. Over the course of ventilator dependency, several changes of rise time might be effected.

The other approach to the problem of adjustable pressure-rise time is what I have called the automatic-shift method. This time the engineers objectively quantify the meaning of the four descriptors for flow gain: gentle, moderate, moderately aggressive, and aggressive. The practitioner selects the descriptor that most closely matches the patient's breathing style, and software analyzes the time-dependent behavior of pressure, iterating the rise-time values until the rise of pressure matches the desired behavior. Whether ventilators will/should receive stick-shift or automatic-shift capabilities cannot be answered at this time. The current literature,[20-22] corroborated in private conversations, suggests that opinion leaders are seriously interested in controlling the rise time of pressure.

Feedback Control

In practice, the equations and approaches that engineers apply to effect feedback control can become quite complex. In simple, straightforward language, feedback control requires that a system be given the capability to measure its own performance and the means to modify its behavior to ensure that observed performance equals desired performance. For many, many years, feedback control has been highly effective in accomplishing sophisticated control through analog design. In recent years, and especially since the availability of cost-effective digital circuitry, feedback control has included a microprocessor in the loop.

With respect to the control of a ventilator's pneumatic subsystem, three control strategies can be found. The Siemens Servo 900C relies entirely on an analog design, whereas the Puritan-Bennett 7200 relies entirely on a digitally based microprocessor design. In the middle is the hybrid design, exemplified by the Hamilton Veolar. Here the flow-delivery valve(s) is(are) tightly controlled by an analog design, whereas the global control of the pneumatic system is under the command of a microprocessor. As yet, no methodology has emerged as the best.

From the standpoint of flexibility of control, the microprocessor-based designs appear to offer some advantages. To appreciate this statement, one must be aware of the role that manufacturing plays in the products one purchases. Manufacturing is the process whereby a given design becomes commonly available. Once a product reaches the manufacturing stage it is considered to be 'frozen,' in the sense that further changes are unwelcome. If a manufacturer believes that a system will require ongoing changes, a microprocessor-based design offers a margin of flexibility. Although software design is just as structured and demanding as hardware design, software can be 'thrown away' without significantly impacting the manufacturing process. One could state, therefore, that microprocessor-based designs provide a high degree of 'upgradability,' whereas analog-based designs provide a lower degree of upgradability.

Enhanced Control through Learning

Microprocessor-based designs have a capacity for learning. Many examples could be given, but one should suffice. Suppose you want your ventilator to deliver exactly 1 liter of breathing gas to the patient's carina (see Reference 24, in which this example first appeared). You therefore select a tidal volume of 1 L. Because your ventilator is a volume-based device (in contrast, say, to a minute-ventilation-based device), you complete your specification by selecting a flow waveform (for example, square) and a value for the peak inspiratory flow (perhaps 60 L/min). Graphically, this example is illustrated in Figure 5. A bit of physics reveals that 1 L will not reach the carina, because in series with the carina are the volume (ie, compliance) and resistance of the patient circuit and artificial airway. At end-inspiration, part of the delivered volume remains in the patient circuit and part reaches the carina. If the volume delivered by the pneumatic subsystem is 1 L, the airway pressure at end-inspiration, $t_{\text{end-inspiratory}}$, equals P_{peak}, and the compliance of the patient circuit (C_C) is known, the volume reaching the carina is given by $V_{carina} = 1000 - (P_{peak} \times C_C)$, expressed in mL. If we choose realistic values, say, 50 cm H_2O for P_{peak} and 4 mL/cm H_2O for C_C,

$$V_{carina} = 1000 \text{ mL} - (50 \text{ cm } H_2O \times 4 \text{ mL/cm } H_2O) = 800 \text{ mL}.$$

Because our smart ventilator computed that 200 mL remained in the patient circuit, the second breath might be set (automatically) to 1200 mL, yielding a P_{peak} of 60 cm H_2O at end-inspiration. Then for the 2nd and successive breaths,

$$V_{carina}\text{, 2nd breath} = 1200 - (60 \times 4) = 960 \text{ mL},$$
$$V_{carina}\text{, 3rd breath} = 1240 - (65 \times 4) = 980 \text{ mL},$$
$$V_{carina}\text{, 4th breath} = 1260 - (67 \times 4) = 992 \text{ mL},$$
$$V_{carina}\text{, 5th breath} = 1268 - (68 \times 4) = 996 \text{ mL},$$
$$V_{carina}\text{, 6th breath} = 1272 - (68 \times 4) = 1000 \text{ mL}.$$

Simple modeling shows that this uncomplicated algorithm converges to yield a V_{carina} equal to V_T (set) in approximately three to five iterations, the variation being due to the values of C_C, C_L (lung-thorax compliance), and resistances of the patient and artificial airway.

Admittedly this algorithm is relatively simplistic, but, in a real ventilator, numerous seemingly insignificant sources of error tend to accumulate and degrade the accuracy of breath delivery. If we endow the ventilator with a microprocessor, along with the capacity to monitor system performance and make the appropriate correction(s) (the principle of feedback control), the accuracy of a subsystem can approach the theoretical value dictated by the combined accuracies of the sensors used to monitor the outputted variable(s).

Corrections Related to the Environment

Microprocessors operating in conjunction with sensors permit a system to self-correct for environ-

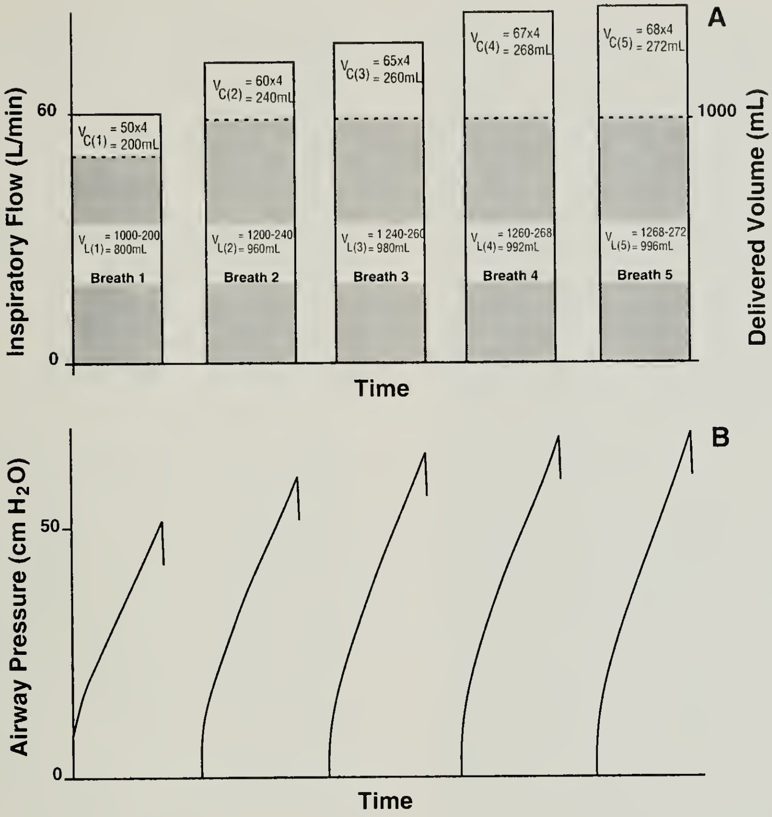

Fig. 5. A depiction of an algorithm that automatically corrects for the compliance of the complete patient subsystem, from the output of the pneumatic subsystem, through the patient circuit, and into the expiratory compartment. With each breath delivery, the algorithm addresses the simple equation,

$$\frac{V_D - V_C}{V_S} = ?,$$

where V_D = total volume delivered on the just-completed breath, V_C = volume in the patient system at end-inspiration, and V_S = set tidal volume. If the answer is < 1.0, the algorithm commands the pneumatic subsystem to increment the delivered volume on the next mandatory breath. If the answer is > 1.0, the next mandatory breath is decreased. An answer of 1.0 means no change. For the algorithm to operate effectively, knowledge of the current value of C_C (compliance of the patient system) is essential. Panel A shows the algorithm's response for

$$\frac{V_D - V_C}{V_S} << 1.0.$$

Note that for V_L to remain constant in the face of changes in C_L, $P_{\text{end-inspiration}}$ will vary (see Panel B).

mental factors. Life can be thought of as existing at the cellular level. Among other essential factors, animal life depends on the body's ability to maintain an adequate P_{O_2} in and around its tissue beds. To quantify this statement, physiologists measure the body's oxygen consumption, in mL/min, under varying states. But the problem is that volume is a highly elusive quantity. Volume depends on temperature, pressure, and the quantities of other gases present—such as water vapor. If Physiologist A is going to be able to communicate quantitatively with Physiologist B, they must develop a common language. This problem was solved when investigators began to express oxygen consumption normalized for gas at standard temperature (0°C), pressure (1 atmosphere), and dry (water vapor removed)—or STPD. Although this choice was logical, given the understanding intended, ordinary humans in their ordinary endeavors inhale ambient air from which the oxygen is extracted and consumed. Thus, our tidal volumes are best conceptualized in terms of body temperature, ambient pressure, and saturated (BTPS), for when our lungs are inflated, the alveolar gas is at body temperature, saturated with water vapor, and referenced to the ambient pressure (see also Reference 24).

To achieve this BTPS normalization, the system design of a ventilator need only be fitted with an absolute pressure transducer. Then, at any altitude and under any barometric condition, the microprocessor simply reads this transducer and applies the current value to its volume calculations.

Special Flow and Pressure Patterns

Functional change is the area wherein the virtues of microprocessor design manifest themselves. With analog design, all circuits must be precisely specified for each and every component. Once specified, verified, and thoroughly tested, the individual components are physically soldered to specially designed circuit boards. The addition of new function requires newly designed circuits and circuit boards—new hardware. This process is both disruptive and costly. Manufacturers generally resist this type of change and, instead, prefer to introduce a new model of the product.

With microprocessor-based designs, change, for the most part, requires new software but not new hardware. The issue is not development time; software design demands the same engineering skills as does analog design. Analog design differs from digital design in one concrete respect. An analog circuit board contains the components that express the function to be performed—say, a square-flow waveform. With digital design, the function resides in software.

Given that the pneumatic hardware meets all specifications required by a new breathing routine, the manufacturer simply issues new software containing the new algorithm. The path to the highly successful Siemens 900C originated with the 900, moved to the 900B, and then to the 900C. Each of these analog-based ventilators exists as a separate model despite many similarities. Knowing that the 900 was introduced in 1972 clarifies why the design centered on an analog design; microprocessor-based electronics, in general, were not cost-effective (for medical equipment) until the end of the 1970s. The Puritan-Bennett 7200 series microprocessor ventilator began its life centered on the Intel 8088 microprocessor. Each new function—pressure-support ventilation, digital communications, respiratory mechanics, flow-triggering, and pressure-control ventilation—was issued as a new software release. Even the S_{pO_2} and display screen options, including waveforms, are centered on software, with some not-insignificant, essential hardware.

A fanciful example at this juncture might prove illuminating. Suppose you obtain a new ventilator whose pressure waveform, in pressure-control ventilation (PCV), measured at the patient Y, always appears as illustrated in Figure 6, Panel A (this result, in itself, would be quite an accomplishment). By experimentation, you record the pressure-time traces at the carina as a function of the size of the ET tube. These traces appear as illustrated in Panel B. Further suppose that your recent experimentation rather convincingly indicates that a large population of patients demonstrates 'improved breathing' when carina pressure reaches the inspiratory-pressure level quickly, say like that illustrated for a #9 ET tube. Subject to flow limitations in the smaller ET tubes, the manufacturer's engineers could develop a controlled, hyperpressurization algorithm that would generate a pressure-time trace at the carina equivalent to the

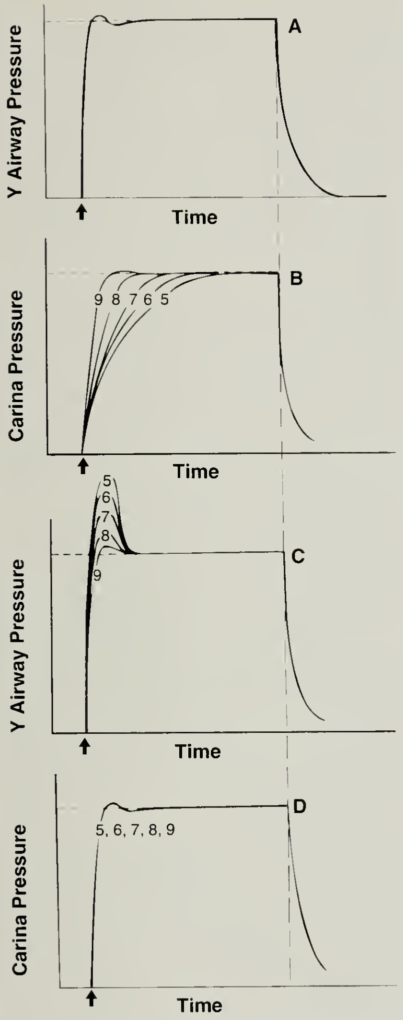

Fig. 6. Global outline of algorithm intended to maintain a fixed pressure-time profile at the carina. Panel A indicates that our hypothetical ventilator includes a smart pressure-control algorithm. Regardless of type of artificial airway, P-T trace at the Y always appears the same. At the carina, however (B), P-T traces display significant differences. Time-to-stable pres-sure lengthens the smaller in diameter (and the longer) the artificial airway is. If the algorithm 'knew' with which artificial airway the patient was intubated, Y pressure could be 'overdriven' (C), yielding (approximately) identical P-T profiles at the carina (D).

#9-ET trace, irrespective of the size of the artificial airway. To achieve this 'enhanced performance,' the practitioner has only to identify the currently installed ET tube. With this knowledge, your enhanced ventilator could produce the unique pressure-time profiles illustrated in Panel C. Again, subject to flow limitations, each pressure-time profile, measured at the carina (Panel D), would appear the same, regardless of the size of the ET tube.

The point of the above discussion is to illustrate the flexibility—the term coined by the manufacturers is 'upgradability'—conferred on a design when it is placed under the control of a microprocessor. This flexibility may entail an incremental cost, but the added cost is trivial, given that a new design must survive in an era in which change is the norm.

Control of Exhalation

As just discussed, an inspection of any new, third-generation, microprocessor-controlled ventilator reveals a pneumatic subsystem considerably more sophisticated and efficient than former designs. Until recently, such a statement would not have applied to a ventilator's expiratory subsystem. The goal of this section is to trace the development of expiratory control, illustrating the current state of this control, developing an understanding of the factors underlying the expiratory 'problem,' and finally, postulating how microprocessor-based control can lead to more efficacious solutions.

A casual but attentive review of the early history of ventilation (as presented in Reference 25) reveals a variety of pre-1950 schemes that were applied to the control of exhalation. These schemes ranged from linkage- or cam-driven sleeves and hard-poppet valves to solenoid-actuated valves. Beginning with the 1950s, numerous 'soft-seat' valves of the diaphragm type appeared, as did valves based on the ping-pong-ball principle. Toward the middle of that decade, one saw diaphragm and flat mushroom valves actuated by pilot tubes. And finally, toward the close of the 1950s and during the early 1960s, the inflatable mushroom valve, piloted by a small-diameter tube connected directly to the output of the ventilator's pneumatic system, became widely adopted. The literature leaves one ambivalent about the reasons behind the apparent acceptance of the inflatable mushroom valve. Perhaps the appeal stemmed from its uncomplicated design, ease of handling, and ready adaptation to a wide variety of manifold designs—or maybe because it was the valve offered on the Puritan-Bennett MA-1.

Popularity aside, other manufacturers introduced different designs, each aimed at correcting an underlying expiratory problem. Very late in the 1960s, Emerson introduced the water-column design. This concept, while providing stable PEEP/CPAP, less tendency to autocycle, and protection against cough-produced pressures, could be somewhat cumbersome. The introduction of the first of the Siemens 900 series ventilators in 1972 brought proportional, active control of exhalation. The constriction of a silicone throat by a scissor mechanism under the control of a stepper motor meant that the valve could assume any position, from open to closed, any time during the breathing cycle. Although much older ventilator designs used solenoid-operated valves, only two positions were possible with them: open or closed.

Perhaps it is reasonable to ask what behavior we expect from an expiratory valve. Four broad requirements immediately come to mind: (1) The valve must seal the patient circuit during inspiration. (2) It must cycle open during exhalation. (3) It must maintain desired levels of PEEP/CPAP. (4) It must not burden the patient's work of breathing. Several factors act to confuse this otherwise simplistic set of expectations. During spontaneous breathing, the ventilator cannot 'know' the precise moment when a patient's respiratory control center will cycle from inspiration to exhalation (see References 26 and 27 for a discussion of the off-switch phenomenon). During controlled or assist/controlled ventilation, this issue poses no burden to the patient because end-inspiration (expiratory valve closed) and beginning exhalation (expiratory valve open) are controlled by a timer within the ventilator. Similarly, but less so, during pressure-supported, spontaneous breathing, exhalation is generally triggered by the inspiratory-flow signal (ie, when declining inspiratory flow equals some fixed percentage of peak inspiratory flow, the ventilator declares exhalation). With a vigorously breathing patient, or with pressure support zero or near zero, exhalation can become a patient-controlled event. In this instance, the ventilator generally looks for a slight rise in P_{aw} of 1.0 to 2.0 cm H_2O above the baseline, which results from relaxation of inspiration in the face of continuing flow or an expiratory effort resulting from the contraction of the abdominal muscles. It should be evident from an appreciation of the actual environment in which the expiratory valve performs that control of exhalation, viewed in the context of the patient-ventilator domain, poses significant challenges.

Repeated laboratory studies[28-30] have demonstrated that none of the valve designs found on ICU-type ventilators prior to the early 1980s performs well when viewed from an ideal standpoint. If ideal means threshold-resistor behavior, all of these valves failed. Mushroom, water column, scissor—all behaved more like flow-restrictors.[28-30] The only design that approached ideal standards was one in which a column of water covered the open, inverted end of an expiratory tube.[28]

Beginning in the early middle 1980s, manufacturers began to develop other designs. All are intended to behave more like threshold-resistors and less like flow-restrictors. Some of the valves use a piloted pressure to control the position of a diaphragm, and some use an electrodynamic motor. (The newly introduced Siemens 300 series ventilator continues to rely on a 'pinch' concept, although the new design has a larger, by approximately 50%, silicone throat and a modified compression mechanism.)

With the current awareness of hyperdynamic inflation and the need to measure and manage it,[31-36] it would appear that expiratory valves must not only behave like threshold resistors but be capable of active control. These requirements, along with others to be discussed later, virtually require that the expiratory valve operate under the control of a microprocessor.

Before I deal with the ramifications of this conclusion, it may prove useful to outline the current understanding of the expiratory process and speculate on the potential for improvement. Just as it is common today to speak of the pneumatic subsystem (meaning an integrated design, operating as a managed ensemble), a modern recognition of the expiratory process requires that it too be framed as a subsystem. For the purposes of this discussion, an expiratory subsystem consists of an artificial airway, a Y-fitting, a 60-in (152-cm) length of expiratory tubing, an expiratory filter (possibly optional, but identified as a Puritan-Bennett SPU filter for this discussion), a one-way check valve (whose function could be satisfied by the design of the expiratory valve), a flowsensor (whose location could be at the patient Y or in the expiratory compartment), and an expiratory valve. Components not studied include in-line water traps (whose flow resistance is a strong function both of the angle included between the inlet and outlet ports and of the streamlining of the bottom of the V where the water trap inlet and outlet ports meet) and heating wires used to eliminate condensate in the expiratory tube.

Because the postulates to follow from this discussion rest on an understanding of the subsystem, selected experiments were conducted especially for this section. The requirement here anticipates a balanced awareness of each of the individual components; later, they will be studied from a subsystems standpoint. The impact of the artificial airway on the patient's work of breathing has received in-depth study.[37,38] Further specific study would add little to our current knowledge.

The first of the selected experiments designed for this section examined the pressure-flow characteristics of 60-in (152-cm) lengths of three common types of adult patient tubing at a PEEP/CPAP of zero. Each tube was connected to one leg of a straight patient Y. Flow entered the Y from the common port, while the remaining leg of the Y was corked. This configuration mimicked the expiratory limb of a standard patient circuit. The three tubes were corrugated reusable silicone, corrugated disposable polyethylene, and smooth bore reusable Hytrel (Smooth Bor, so-called because the inner surface is smooth rather than corrugated). The results of these flow-ΔP trials appear in Figure 7. Note that each tube was studied in each of three configurations: straight, coiled in a circle whose circumference equaled the length of the tube, and S-shaped.

These data demonstrate a strong influence of curvature on ΔP for the corrugated silicone and polyethylene tubes. For the corrugated silicone tube at flows of 100 L/min and above, the circle and S-shapes increase the ΔPs in the ranges of 34-38% and 40-44%, respectively (taking the ΔP value for the straight configuration as a standard). For the corrugated polyethylene tube at flows of 150 L/min and above, the circle and S-shapes increase the ΔPs in the ranges of 61-62% and 75-70%, respectively (straight configuration equals standard). In contrast, the smooth bore tube at flows of 100 L/min and above exhibits only a 10-14% gain in ΔP, which is largely uninfluenced by the configuration inducing the increase in resistance—circle or S-shape. Irrespective of the configurational shapes chosen for the silicone and polyethylene tubes—circle or S—each tube type exhibited marked differences in ΔP across all flows.

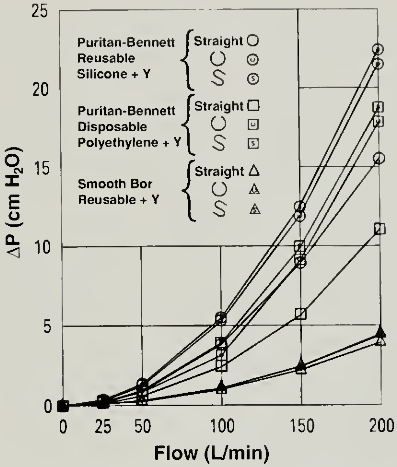

Fig. 7. Flow-pressure-drop (ΔP) relationships measured for three different 60-in (152-cm) lengths of adult patient-circuit tubing, each formed into three 'standard' configurations—straight, circular (one loop), and S-shaped. Gas entered the inlet end of each tube through the common port of a patient Y. The other port of the Y was sealed with a cork. The outlet of each tube opened to the room.

Beginning at flows of 50 L/min, the disposable polyethylene tube exhibited a ΔP at least 150% greater than the value for the smooth bore tube (taking the circular shape for comparison). By contrast, the reusable silicone tube registers at least a 275% increase. At flows of 200 L/min, these percentages increase to at least 275% and 375%, respectively. The marked impact of tube material and configuration on flow resistance will be discussed later in connection with an examination of the data presented in Figure 11.

The next component studied was the flow-sensor. Four elements were tested, and their pressure-flow data appear in Figure 8. Each element was removed from its parent ventilator. [Note: for this part of the study, the four ventilators identified earlier were again called into service. Each is a third-generation machine. Their expiratory compartments were examined component-by-component and then in various assembled configurations. To keep track of each of these ventilators and its components, the following assignments were made: Ventilator 1 (V1), O-O, Ventilator 2 (V2), □-□, Ventilator 3 (V3), Δ-Δ; and Ventilator 4 (V4), ∇-∇. These assignments apply to Figures 8-11 and 13.]

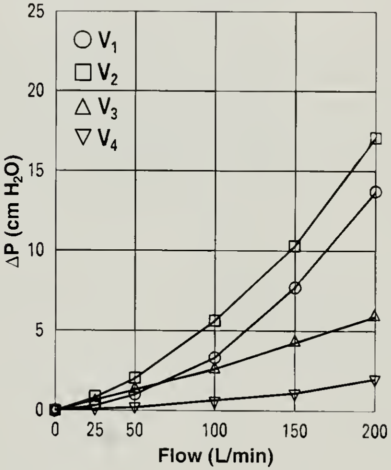

Fig. 8. Flow-pressure-drop (ΔP) relationships measured for four isolated flow elements removed from third-generation ventilators. PEEP/CPAP for these tests was set to zero.

The data shown in Figure 8 indicate a substantial difference in the ΔP-flow profiles for each flow-sensor. At the lowest flow, 25 L/min, the ΔPs' range was approximately 0.1-0.8 cm H_2O. This eight-fold factor manifests itself again at 200 L/min. Here the lowest ΔP measures approximately 2.0 cm H_2O and the highest, approximately 17.1 cm H_2O. At flows between 0 and 50 L/min, the order of AP is V2 > V3 > V1 > V4. At flows between 100 and 200 L/min, the order is V2 > V3 > V1 > V4.

Pressure-flow data for three of the four expiratory valves appear in Figure 9 (the valve from V4 could not reliably be separated from its parent subsystem). Again, the data show substantial differences among the three designs. At 25 L/min, the ΔPs' range was approximately 0.2-0.7 cm H_2O and at 200 L/min, approximately 3.3-8.3 cm H_2O. (Note that the ΔP values for V2 and V3 overestimate the true values because the check valves for these two units were not separable from their expiratory valves.) As seen in this figure, the order of the ΔPs at each flow was not uniform, as it was for the ∇s. At 25 L/min, the order is V2 ~ V3 > V1. At 50 L/min, the order becomes V3 > V2 ~ V1. But above 100 L/min, the order shifts to V3 > V1 > V2 . Although the exact reasons for the shift in the ΔP-flow profiles are not pertinent to the goal of this paper, it should be noted that the ΔP-flow profiles strongly depend on the physical design of the valve. Factors such as the orientation of the di-

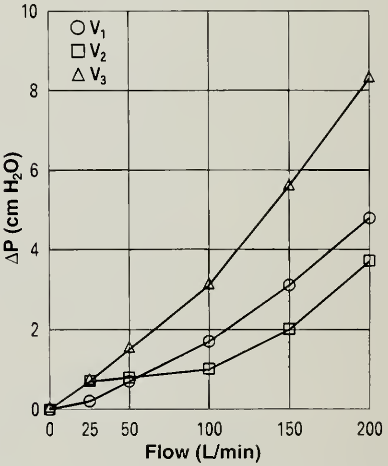

Fig. 9. Flow-pressure-drop (ΔP) relationships measured for three isolated expiratory valves removed from third-generation ventilators. For technical reasons, the valve from Ventilator 4 was not studied. Note that the valves from Ventilators 2 and 3 had integral, one-way valves; hence, their ΔP values slightly overestimate the true values for the valves. PEEP/CPAP for these tests was set to zero.

aphragm and whether it is weighted or guided introduce nonlinearities into the ΔP-flow relationship.

At this point, the pressure-flow relationships for the assembled expiratory compartments were studied. The ΔP-flow profiles appear in Figure 10. At 25 L/min, the ΔPs' range is approximately 1.0-2.0 cm H_2O and at 200 L/min, approximately 18.3-28.3 cm H_2O. The order of the ΔPs at 25 L/min is V4 > V2 ~ V3 > V1. At 50 L/min, the order becomes V3 > V2 ~ V1 > V4. And at 100 L/min, V1 ~ V3 > V2 > V4. Finally, at 150 L/min and above, V1 > V3 > V2 > V4. (Note: for technical reasons, the data for V2 slightly overestimate [5% at most] the true values.) The ΔP-flow profiles for the complete expiratory compartments (flowsensor, check valve, and expiratory valve, plus all interconnecting fittings) thus generally exhibit the summation of the profiles of the individual components.

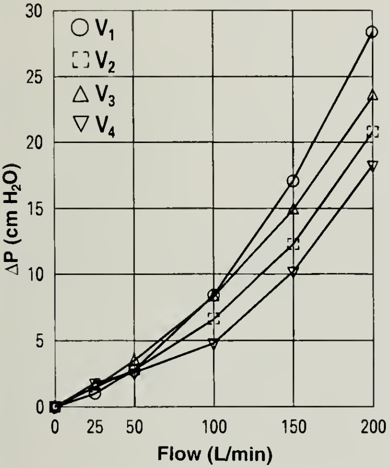

Fig. 10. Flow-pressure-drop (ΔP) relationships measured for the four intact expiratory compartments of the four third-generation ventilators. Each compartment was studied in the 'as-shipped' configuration. For technical reasons, the ΔPs for Compartment 2 are best estimates rather than true values. PEEP/CPAP for these tests was set to zero.

Finally, all of the components common to the standard expiratory subsystem were interconnected and their pressure-flow profiles were measured. Each circuit consisted of the corked Y, the 60-in (152-cm) straight length of smooth bore tubing, an elbow fitting to connect the expiratory tube to the expiratory compartment (if normally part of a system), a SPU filter (same one each time, even when it was not part of a system), a flowsensor, a check valve, and an expiratory valve, plus all normal interconnecting fittings. Figure 11 displays the pressure-flow profiles. At 25 L/min, the ΔPs' range is approximately 1.3-2.0 cm H_2O, a spread of less than 1 cm H_2O. At 200 L/min, the ΔPs' range is approximately 24.9-29.4 cm H_2O, a spread of not more than 6 cm H_2O. Note that at flows of 25 and 50 L/min, V1 exhibits the lowest ΔP, and at 100 L/min, V4 is lowest. But at 150 and 200 L/min, the measured ΔPs for V2 were the lowest.

Up to this point, two factors stand out: first, the performance of a given expiratory valve only fractionally determines total (expiratory) system performance; and second, the separation between the lowest and highest ΔPs at any flow (Fig. 11) above

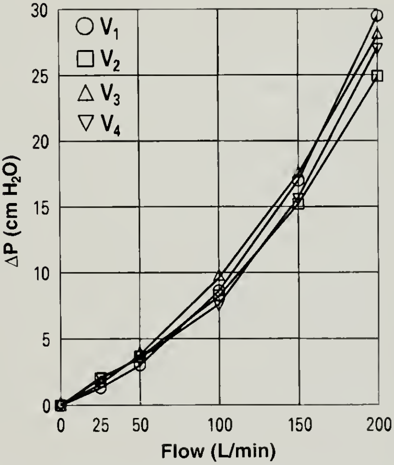

Fig. 11. Flow-pressure-drop (ΔP) relationships measured for each of the four expiratory compartments connected to the straight length of Smooth Bor patient-circuit tubing (with corked Y) identified in Figure 7. For these tests, all compartments were fitted with the same Puritan-Bennett SPU expiratory filter. With the addition of an appropriate artificial airway attached to the patient Y, the components studied during this part of the test comprise the minimum expiratory subsystem as defined in the text. PEEP/CPAP for these tests was set to zero.

50 L/min would be more than offset by using disposable, corrugated polyethylene tubing or reusable, corrugated silicone tubing. Stated another way, any advantage that might be gained by the expiratory subsystem with the lowest flow resistance would be overwhelmingly offset by polyethylene or silicone tubes (compared to smooth bore), severe bends, heated wires, water traps, and water in the lazy loops. On the other hand, optimization of all factors that contribute to flow resistance in combination with the expiratory compartment with the lowest flow resistance yields the superior expiratory subsystem.

Before I focus the topic of control of the expiratory process back to microprocessor technology, the dynamic behavior of our representative, third-generation expiratory systems should be mentioned briefly. The dynamic model for this phase of the study (Fig. 12) consisted of one half of a Michigan Instruments Training Test Lung (TTL) connected to an electronically switched, large-bore, piloted valve by an approximately 12-in (30-cm) length of standard adult-size tubing. To the outlet of the piloted valve was connected an ET tube, the standard expiratory circuit, and, lastly, the expiratory compartment under study. The expiratory compartment of each ventilator contained only the factory-fitted equipment, except for the inclusion of the same Puritan-Bennett SPU disposable filter, even when such a filter was not part of the ventilator's standard set-up. (Note that for this experiment the ventilator itself was bypassed; only the expiratory compartment was connected to the TTL model lung.) A pressure tap at the simulated carina position completed the model. Pressure-time traces were run for each trial on a Gould Model TA2000 digital strip-chart recorder. Calibration was adjusted to 50 cm H_2O full scale (20 cm), and the paper speed was 100 mm/s. Each trial began with the lung filled. In quick succession, the chart recorder was switched on and the piloted valve was switched open. Thus, this model simulated a patient who inhaled a tidal volume of 1 L and then exhaled passively.

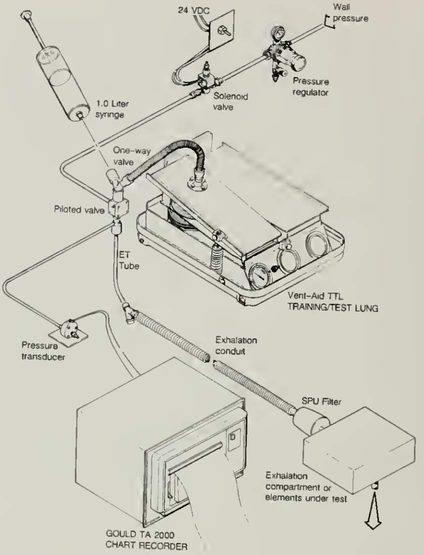

Fig. 12. Illustration of the dynamic model used to derive ΔP-time traces during a passive, compliance-driven exhalation from a Michigan Instruments Training Test Lung (TTL; only one chamber active) filled previously from a 1.0-liter syringe. A mechanical stop in the syringe ensured that all volumes were identical. The leak tightness of the lung, interconnecting tube, and piloted valve was verified by observing the pressure in the lung following the injection of 1.0 liter into the lung chamber. Pressure declined only fractions of a cm H_2O during the interval required to start the recorder and initiate an exhalation. Pressure during exhalation was read from a tap placed at the 'carina' position. In this model, only three elements were varied: (1) the compliance of the TTL (0.050 or 0.030 L/cm H_2O), (2) the size of the endotracheal tube (#9 or #7), and (3) the expiratory compartment (V1, V2, V3, or V4). The configuration of each expiratory subsystem under test consisted of all standard elements (flow-sensor, water trap, tubes, etc) plus the same 60-in (152-cm) straight length of Smooth Bore tubing and Puritan-Bennett SPU expiratory filter, whether or not this item was standard.

Figure 13 shows typical dynamic expiratory traces for each of the four ventilators (C = 0.30 L/cm H_2O, #7 ET). Also included for comparison are the control traces recorded identically, except for the uncoupling of the ventilator. Repeatedly recorded traces of any type exhibited superposition within the limits of the width of the tracing (Panel A). Note that the control trace (without any expiratory compartment) indicates that the time for pressure to decline from its initial value (measured inside the lung, approximately 33 cm H_2O) to 5% of initial value (e^{-3}) equals approximately 940 ms. (See Reference 39 for a thorough development of

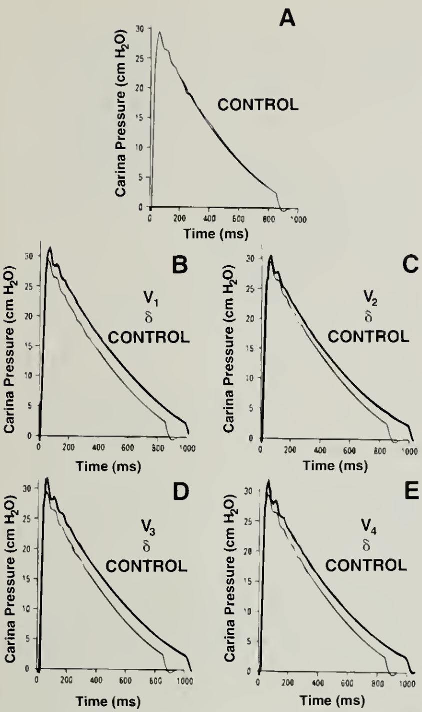

Fig. 13. Dynamic, expiratory pressure traces for each of the four expiratory subsystems, including the control trace recorded identically but minus a ventilator. Compliance was set to 0.030 L/cm H_2O and the ET was a #7. Prior to initiation of the exhalation, the lung chamber was filled from a 1.0-L syringe. Panel A exhibits trial repeatability by showing the superposition of two separate (control) traces. Panels B, C, D, and E show a control trace along with a trace recorded with one of the four expiratory compartments included as an element of the expiratory subsystem. Thus the difference between a control trace and an experimental trace depends only on those elements included in each ventilator's expiratory compartment, as detailed in Figure 10.

the dynamic behavior of the respiratory subsystem.) This statement is important. It means that for any real expiratory subsystem (including an expiratory compartment), the minimum expiratory time cannot be less than 940 ms (configuration defined above, including PEEP/CPAP = 0, a #7 ET tube, e^{-3} factor). This minimum time extends to approximately 1.25 s with a 98% decline (e^{-4} factor) in pressure. The data in Table 1 summarize the analyses of all trials conducted with the dynamic model.

Table 1. Estimated Time for Carina Pressure To Decline from Pre-Exhalation Value of Chamber Pressure (≈ 32 cm H_2O, Measured) to e^{-3} (or 5%) of that Value (≈ 1.6 cm H_2O)*

	C = 0.030 L/cm H_2O		C = 0.050 L/cm H_2O	
Configuration	#9 ETT	#7 ETT	#9 ETT	#7 ETT
Control	0.69†	0.94	0.83	1.17
Complete Exhalation System	0.79-0.86	1.09-1.12	1.06-1.113	1.38-1.46

* C = compliance, ETT = endotracheal tube.
†Values are in seconds.

One way to understand the data from the dynamic model is to compare each of the pressure-time traces recorded with a given ventilator to its control trace (Figure 13, Panels B, C, D). This comparison generated four tables in which the four control curves (C = 0.50 and 0.30 cm H_2O, ET = #7 and #9) were subtracted from the four matching curves generated with each of the four ventilators. The results of one such exercise appear in Figure 14. All of the difference curves demonstrate a similar pattern. A few milliseconds after exhalation began, the experimental curves measured little more than 1.0 cm H_2O away from the control curve. Approximately 600 to 700 ms after exhalation began, the experimental and control curves exhibited the maximum separation, between approximately 2.1 cm H_2O for the least burdensome expiratory system and 2.7 cm H_2O for the most burdensome expiratory system. At the 100-ms mark (after the initiation of exhalation), the order of ΔPs was V1 ~ V3 > V4 > V2. From 200 to 700 ms, the order became V3 > V1 > V4 > V2. At the next measurement time, the order shifted to V3 > V1 > V2 ~ V4. By the 900-ms mark, the order shifted again, becoming V3 > V1 ~ V2 ~ V4. After 1000 ms, when the data can only be estimated, the probable order of ΔPs was V3 ~ V4 > V1 ~ V2. For the case of passive exhalation and zero PEEP/CPAP, all four ventilators achieved an e^{-3} decline in (carina) pressure by approximately the 1.1-s mark.

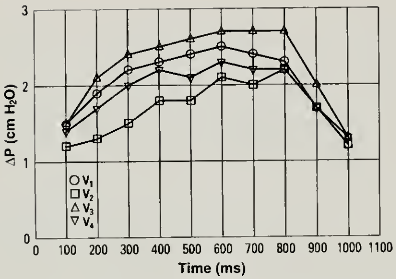

Fig. 14. Dynamic, difference-ΔP profiles plotted from data obtained for each of the four expiratory subsystems. Difference ΔPs were calculated by subtraction of the control ΔP-time profiles recorded *without* the expiratory compartments from the matching, experimental ΔP-time profiles recorded *with* the expiratory compartments attached (see Fig. 13). Exhalation was driven by the volume-loaded (dynamic), TTL model as illustrated in Figure 12. Data points for the 1.0-second mark are estimates made by extending by one interval the semilogarithmic plots of the raw data. PEEP/CPAP for these tests was set to zero.

Although a detailed analysis of the dynamic results has little bearing on the goal of this paper, some qualitative statements appear relevant. The ΔP-flow profiles shown in Figure 11 (all common components from and including the patient Y to and including the individual expiratory compartments) suggest that V1 exhibited the lowest ΔPs between 25 and 50 L/min, whereas V2 exhibited the lowest values between 150 and 200 L/min (and V1 registered the highest value at 200 L/min). At 50, 100, and 150 L/min, the highest ΔPs were registered by V3. All of the dynamic, differential ΔP curves were qualitatively similar to those shown in Figure 14. During the early part of exhalation, when high flows dominated, V3 generally exhibited the highest differential values and V2 the lowest. Toward the end of the exhalation, when flows were reduced, the lowest differential values were generally registered by V1. The point of importance is that the shortest expiratory time results from the dynamic interaction of all of the elements comprising the expiratory system.

With a qualitative appreciation for the behavior of current expiratory systems operating at zero PEEP/CPAP, it is possible to develop an understanding of an expiratory system running under microprocessor control. In the face of the same tidal volume or active inspiratory pressure (measured from PEEP/CPAP to the "target" pressure during pressure-control ventilation [PCV]), the selection of a level of PEEP/CPAP causes the end-inspiratory pressure in the lungs to increase by a value approximately equal to the setting for PEEP/CPAP (more or less equal if volume ventilation is active, and equal if PCV is active). This result means that the pressure gradient from lung to atmosphere increases, whereas the gradient from lung to PEEP/CPAP remains approximately the same. If the conventionally controlled and pneumatically piloted expiratory valve retains its same efficiency with and without PEEP/CPAP, which most likely does not happen,[28-30] the expiratory pressure-time profile remains essentially the same regardless of the PEEP/CPAP value. Because of inefficiency, expiratory time usually increases as PEEP/CPAP increases. If, however, the ventilator could take advantage of the lung-to-atmosphere pressure gradient, instead of being constrained to the lung-to-PEEP/CPAP gradient, exhalation with finite PEEP/CPAP could be more efficient than (or at least as efficient as) exhalation with PEEP/CPAP equal to zero. To achieve this goal, the air chamber behind the diaphragm (Fig. 15) is effectively referenced to atmospheric pressure early in the expiratory event, then smoothly referenced back to the effective PEEP/CPAP pressure at precisely the right time to re-establish the desired PEEP/CPAP setting.

The type of control just described for the expiratory valve is termed "active." Active implies that the behavior of the valve under all anticipated conditions is specified by an algorithm whose commands are executed via a control system (for the purposes of this paper, read: microprocessor). Active control generally follows one of two schemes: the first entails manipulation of the pressure in the air chamber behind the diaphragm (Fig. 15, Inset B), and the second uses a proportional, linear solenoid directly or indirectly coupled to the diaphragm. Exhalation in the Dräger IRISA typifies active pneumatic control, whereas exhalation in the Hamilton Veolar and Amadeus typifies active control managed by an electrodynamic motor. Operationally, these active valves are controlled by an al-

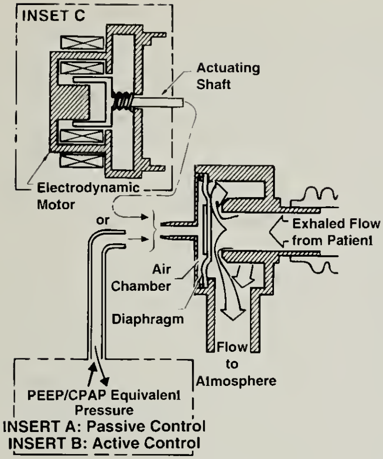

Fig. 15. An illustration of a diaphragm-type expiratory valve. Diaphragm positioning generally follows one of three strategies: (1) maintenance of a specified, PEEP/CPAP-equivalent pressure in the air chamber (passive, pneumatic control; see Inset A), (2) algorithmic modulation of the chamber pressure between the PEEP/CPAP-equivalent value and a lesser value, thereby improving expiratory efficiency when PEEP/CPAP is finite (active, pneumatic control; see Inset B), and (3) algorithmic positioning of the diaphragm via an electrodynamic motor (active, direct control; see Inset C). If the electrodynamic motor and the diaphragm are directly coupled (in contrast to physically uncoupled), all positions of the diaphragm must be specified by the control algorithm. In the uncoupled design, the force of exhalation opens the valve, but closure is controlled by the electrodynamic motor.

gorithm that monitors breath-timing and airway pressure.

Although the design of such an active control algorithm may appear straightforward, the task presents considerable challenges, especially when PEEP/CPAP assumes finite values. Unlike the inspiratory event, which is controlled by valves regulating flow derived from an infinite source and stable pressure, the expiratory event begins with a finite source (volume), driven by a diminishing pressure. Thus, inspiration resembles the take-off of an airplane with full control of power, while exhalation resembles the landing of an airplane 'dead stick' (that is, with no power). Exhalation mimics a quasi-explosive event. The opening force acting on the diaphragm may be so great that too much gas escapes from the patient circuit before the PEEP/CPAP-equivalent pressure becomes established. This action may lead to a sub-PEEP/CPAP pressure and, hence, autocycling. To control this unwanted side effect, valve engineers restrict the rate at which the diaphragm can be driven off of the valve seat. Many times, the effect of such restraints, although successfully inhibiting undesirable behavior, adds to the flow resistance of the expiratory valve. Today's challenge to ventilator manufacturers is that of designing a cost-effective expiratory valve, maximizing the desirable characteristics and minimizing the undesirable ones. Such a task is well suited to microprocessor design.

Harmonization of Inspiration and Expiration

Once the expiratory valve is placed under microprocessor control, undesirable pressure phenomena occurring during inspiration (eg, pressure overshooting due to coughing) can be relieved by pulsing open the expiratory valve. Optimum performance of APRV (airway pressure release ventilation), for example, reportedly depends on minimizing the interval during which the high level of CPAP is released to the low level and then re-established at the high level.[40,41] Exhalation in its broadest context is no longer seen as an isolated event. Rather, the expiratory valve and the inspiratory valve(s) operate in harmony to maximize the efficiency of the inspiratory and expiratory events. If the analogy of a dead-stick (power-off) landing of an airplane adequately conveys the image of exhalation without active control, then exhalation integrated with the pneumatic subsystem is analogous to landing an airplane under controlled power.

An excellent example of the integration of the expiratory valve into the more global aspect of ventilator function, all under microprocessor control, is the programmed, automatic measurement of intrinsic PEEP (PEEP$_I$). East and coworkers[42,43] have designed a prototype computer program for the maintenance of a desired level of PEEP$_i$, the new scheme eliminating the need for set PEEP. In the near future, actively controlled expiratory valves, acting in concert with the pneumatic subsystem, will become an indispensable component of sophisticated ventilator design.

The User Interface

Clinicians seem to approve of sophisticated equipment as long as it is easy to use. (See Reference 44 for an interesting discussion of the requirements for sophisticated user interfaces.) At first glance, this statement appears inconsistent; yet upon introspection, an understandable logic emerges. As the 1970s drew to a close, few people realized that the proliferation of the personal computer lay just around the corner. Specialty micro- and mini-computers were in the hands of knowledgeable users, but the average person 'made do' with paper and pencil or possibly a typewriter. Software and affordable price then made the personal computer available to nearly every desk. Software buried the complex tasks behind a 'screen' of simplicity. Although the equivalent breakthrough has not yet occurred in ventilator design, the application of 'friendly' software to the user interface cannot be too far away. While one could argue for an analog-based gas-delivery system, few would reject the case for a microprocessor-based user interface. The ease-of-use issue extends beyond the question of knobs versus keys or how information is communicated within or outside of the ventilator (see Reference 7 for a short discussion of this subject). For at least the last 15 years, a 'standard' adult ICU ventilator has been expected to offer A/C, SIMV, and CPAP with pressure-triggering and volume-based mandatory breaths. More recently, clinicians have come to expect pressure support and possibly pressure control. Forgetting the conventional for the moment, suppose your next ventilator offered, in addition to the standard features, flow-triggering, pressure-support ventilation, pressure-control ventilation (and perhaps volume-priority pressure ventilation), airway pressure release ventilation,[40,41,45-47] proportional assist ventilation,[48] mandatory minute ventilation, programmable inspiratory and expiratory pause, respiratory mechanics and weaning parameters, S_{pO_2}, integrated smart end-tidal CO_2, integrated metabolics, work of breathing, real-time blood gases,[49-52] not to mention graphics (waveforms and trending), smart alarms, advisory closed-loop ventilation,[53-55] closed-loop ventilation based on high-level intelligence,[56-58] and, finally, digital communications (bedside, remote, and telemetered)—as well as the capability to ventilate neonatal, pediatric, and adult patients. Such a system could well be inundated with knobs, levers, buttons, and specialty displays. Not only would a conventional user interface cost nearly a fortune to manufacture, but only a dedicated optimist would be inclined to master the system.

Only through thoughtful software design (implied microprocessor control) can such a system be managed. With careful analysis of each function and its relationship to the whole, the apparent confusion of functions begins to sort itself out—SIMV but not CMV, CPAP, APRV, or MMV; flow-triggering but not pressure-triggering; PAV but not PSV; PCV but not volume-based ventilation; and adult ventilation but not neonatal or pediatric. The selection of a few functions obviates others.

Coding of the logic tree in software permits the ventilator to know how to configure itself once the operator identifies his or her selections. Microprocessor control lends itself extremely well to this type of situation. What the clinician sees and interacts with are just those subgroups of functions that enable him or her to control the ventilator. Even within the subgroups, many functions—sigh and apnea ventilation, for example—can remain out of sight, recallable with a single, simple command.

By what means does the clinician manage such a system? No single best solution exists. Today's designer has many technologies from which to choose: mouse, track ball, rotary encoder, pistol-grip handle, soft key, and touch screen, to name the most common. Continuing study and experimentation will be required before an optimal solution emerges. One can suggest that no single technology will satisfy all requirements. Each specific environment demands its own solution.

Display of Patient Data

With software working in conjunction with powerful, fast microprocessors and seemingly unlimited memory, how can limits be set on the processing and displaying of data? Because virtually all possibilities can be implemented, does that mean they should be? One approach to the problem of data overload is to review what the clinician needs to see if he or she is to make intelligent decisions. Data for the sake of data must be rejected. The minimal requirement demands that data be translated into information, and the information must be relevant to patient care.

In the past, information consisted of numbers: for example, set V_T, 500 mL; measured exhaled V_T, 480 mL; assist-control respiratory rate, 18 breaths/min; peak inspiratory pressure, 55 cm H_2O; mandatory minute ventilation, 8.64 L. When most ventilator fleets in the United States consisted of MA-1s and Bear 1s and 2s, sophisticated display of data lacked urgency. Recent clinical studies suggest that all was not as simple as assumed.[11,59] Figure 16, taken from Reference 59 (1986), illustrates the conceptual problem. The solid P-V trace in the upper panel results from a controlled, square-waveform, mandatory breath (passive patient). Conventional wisdom believed that when the patient triggered the mandatory breath, the P-V trace would appear somewhat similar, as suggested by the dashed trace (upper panel). With the recent proliferation of pressure-time (P-T) waveforms, most clinicians now recognize that an assist-controlled P-T waveform differs significantly from a controlled P-T waveform. The impact of this discrepancy on a patient's work of breathing, however, was previously little appreciated. By superimposing a control curve and an assist-control curve, one can resolve the work done by the patient from that done by the ventilator (refer to the bottom panel in the figure). Marini et al[11] found that the work performed by patients during 100% assist-control breathing was far from insignificant, measuring approximately 40% of the work observed during 100% spontaneous breathing. Perhaps more interesting was the finding that the work per volume of each assist-controlled breath increased as the percentage of assist-control diminished (the decline in assist-control breathing being made up by an increase in the percentage of spontaneous breathing).

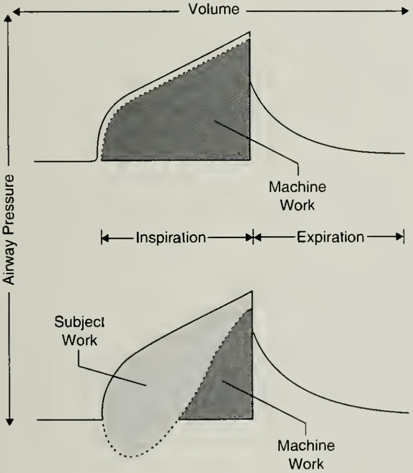

Fig. 16. Illustration of assumed versus actual work during volume-based, assist-control ventilation (square flow waveform). Shown in the upper panel are P-V traces resulting from a controlled breath and an assisted breath. Clinicians generally believe that both traces would appear as illustrated—nearly identical. Recent analysis[59] suggests that the P-V trace for an assisted breath has the appearance shown in the bottom panel. Superposition of the controlled and 'actual' assist-control traces allows the partition of machine and patient work. (Reprinted, with permission, from Reference 59.)

This discussion suggests that the P-T waveform reveals specific information not reducible to simple numbers (at least not without special techniques involving placement of an esophageal balloon). Suppose you observed a patient on assist-control ventilation whose P-T waveform had the appearance of Trace b in Figure 17. The trace clearly evidences the trigger effort and the subsequent smooth rise to peak pressure at end-inspiration. Some time later, you check up on your patient; his or her P-T waveform now looks like that labeled c. Having gained a facility for reading waveforms, you correctly interpret Trace c as indicating flow starvation. After some experimentation with increased values for peak inspiratory flow, you are able to repair the sag in Trace c, restoring it to the shape illustrated by Trace b. Suppose further that you could select another triggering strategy for your ventilator, which yielded Trace d. You might justifiably feel a sense of satisfaction with your newly mastered skills.

Compared with the strategies for monitoring the assist-controlled patient, the situation becomes considerably more complex when the patient begins to experience significant spontaneous breathing. Unlike the patient on assist/control (volume-based)

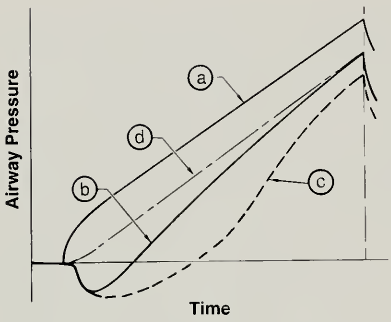

Fig. 17. Depiction of pressure-time traces recorded at the patient Y during volume-based, assist-control ventilation. Trace a illustrates the P-T profile for a machine-controlled breath. As the patient begins to exert inspiratory effort, the P-T profile will shift. Trace b illustrates an acceptable balance between patient work and ventilator work. Trace c suggests that the patient's demand for flow and the flow output of the ventilator are poorly matched. Trace d illustrates an example in which the demand for flow is instantaneously met and demanded flow and delivered flow are well matched throughout the remainder of the breath.

ventilation—in which the flow-time profile is completely specified by the clinician, leaving the patient in control only of respiratory rate—the spontaneously breathing patient controls all facets of the minute-ventilation and flow profiles (with $\int \dot{v}dt$ = volume) as well as respiratory rate. Until the patient recovers from the underlying problem that caused ventilator dependency, the clinician must make effective use of the ventilator to manage the patient's work of breathing. Because work equals pressure × volume, the external application of pressure, via the ventilator, effectively allows the externally applied work to harmonize with patient-generated work. While the current literature now offers many excellent papers dealing directly with pressure support and its ability to manage work of breathing,[60,61] assessment of the adequacy of the patient's support still remains largely an art rather than a science.

The quantification of harmony (as in patient-ventilator harmony) presents innumerable difficulties. Because of the human brain's ability to process complex information via pattern recognition, the graphic display of information may circumvent many of the ambiguities inherent in the design of interpretative algorithms. Long before ECG rhythms were processed by software, the meaning of irregularities was deduced by the process we humans define as pattern recognition. The ability of a ventilator to display waveforms has become a customer demand. My experience, however, is that most clinicians do not yet feel comfortable with the task of (waveform) pattern recognition. MacIntyre and Hagus recognized this deficiency, publishing their monograph on waveforms and their interpretation.[62] For waveform analysis to reach the genuinely practical level, manufacturers will most likely have to distribute sophisticated learning materials (see Reference 63 for a new addition to the list of waveform-training booklets).

Once a system design includes one or multiple microprocessors (and associated memory), data manipulation and processing are readily added. These features do incur additional cost, owing to specific hardware and software requirements. The cost will be incremental, however, because of the ready availability of the raw data. Microprocessor electronics (a convenient term that includes microprocessor, electronics, and software) excel at data processing, storage, and manipulation. If the basic ventilator design includes a screen, as in the Bear 5 and the Adult Star, a means of display already exists. Perhaps not surprisingly, the manufacturers of anesthesia machines have taken a temporary lead with respect to the use of display screens and the presentation of complex data (Fig. 18).

As clinicians who practice respiratory care and ventilator management (physicians, therapists, and nurses) become more adept at managing microprocessor-based equipment, manufacturers can take the risk of introducing more sophisticated devices. Several factors common to the practice of anesthesia, but absent from the practice of respiratory care, seemingly have aided the development of smarter, ergonomically organized, microprocessor-controlled, anesthesia machines. Such driving forces might include the rising proliferation of ancillary equipment essential to the practice of anesthesia, the rising number of mishaps in the operating theater, the rising cost of liability, a commonly trained, uniform class of device operators—the anesthesiologists themselves—and a favorable relationship between risk reduction and incremental

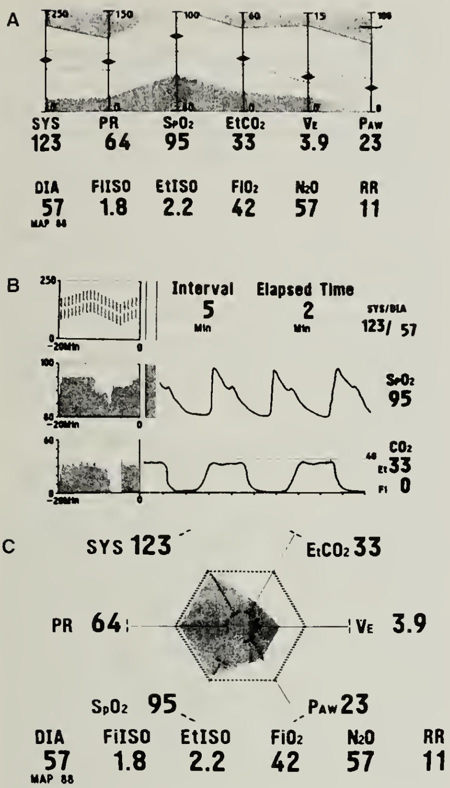

Fig. 18. Examples of information displayed on the face of a configurable 'screen.' Note the balance between numeric and analog data, real-time and trended data. With the proliferation of screens—primarily video, liquid crystal display, and electro-luminescent—clinicians can expect to see a rapid rise in the intuitive display of information. (Permission to print granted by Ohmeda, A Division of BOC Health Care Inc.)

cost (see Reference 64 for papers). Compounding the issue of sophistication and ventilator design are several divergent factors, such as the multiplicity of user education, training, and background, a common user outlook that encourages sophistication but not at the expense of complexity, and the perception on the part of the user that sophistication may be outstripping benefit. Notwithstanding these obstacles, ventilator manufacturers and clinical researchers, working individually or jointly, evince concerted efforts in the direction of sophistication and ease of use.[65,66]

Data Transmission to Remote Locations

Once a ventilator's microprocessor electronics have captured, processed, and manipulated data for use within the device, distribution of this information to remote locations can be effected for a relatively modest incremental cost (see Reference 67 for a balanced discussion of the issues and concerns regarding transmission of digital data from ventilators to computers and printers). Puritan-Bennett's 7200 ventilator first offered a digital data output in 1985. This feature is now common to all third-generation, microprocessor-based ventilators. Although not microprocessor-based, the Siemens 900C acquires this capability with the addition of their 990 Digital Communication Module. The newly introduced Siemens 300 series ventilator is based on a multiprocessor design with ample input-output, digital communications capability. The lack of a communication protocol common to the medical-device industry, as well as the lack of standardized receiving modules, inhibits the availability of potentially useful information to the clinician. A Siemens monitor will receive information only from a 900C or SV300, a SpaceLabs monitor will link to a 7200,[68] and Hewlett Packard now offers links to the 900C and 7200—but users who lack these specific configurations receive no benefit. The MIB (medical information bus) offers some hope, but manufacturers have not uniformly endorsed the protocol.[69] Although the manufacturers of monitors may be willing to supply communications modules for the more popular ventilators, manufacturers with smaller market shares will be left out unless a uniform standard is adopted. Clinical departments that early on adopted a strong stand in favor of remote data display simply wrote their own software (personal communication, Dr Graziano Carlon, Sloan-Kettering Institute, New York NY, December 1990).

Whatever the current level of frustration regarding the lack of common communication protocols, I perceive a strong demand for this capability. Given progress in this direction, some potentially interesting possibilities emerge. With continuing pressures for cost containment, one can envision not only remote display of data at central stations, but the transmission of specific information to receiving devices worn or carried by on-duty practitioners. These receivers may resemble pagers or powerful hand-held computers such as those sold with Puritan-Bennett's Clinivision data-management system.

Although beyond the scope of this paper, the issue of remote monitoring raises many concerns regarding the impersonalization of patient care. As the possible becomes feasible, society and the medical community must discover ways to explore and resolve pressures that tend to erode the practitioner-patient relationship.

Ventilator Alarm Strategies

A ventilator not only needs to offer all of the requisite modes and controls for uniquely tailoring gas delivery to every patient, but, in addition, a ventilator must be capable of identifying when it, the patient, or the ventilator-patient relationship is jeopardized. In parallel with simplification of the operator interface, microprocessor electronics has the potential to enhance and improve the current offering of alarm strategies. In this section, I review the development underlying third-generation alarms and highlight some of the ways in which microprocessor electronics could lead to more satisfying alarm strategies.

Brief Developmental History

The first ventilator, the Fell O'Dwyer apparatus introduced in 1888, very closely resembled a modern-day, foot-operated basketball pump. It had no alarms. In time, early ventilators were fitted with a pressure gauge to monitor airway pressure. Puritan-Bennett's popular MA-1 contained only two alarms.

Today the Siemens 900C offers eight alarms, but they are not organized into a unique alarm section. Puritan-Bennett's 7200, introduced in 1983, now has 15 alarms, excluding those associated with S_{pO_2}, and 7 of the 15 are settable. But still, in SIMV the alarms for mandatory and spontaneous tidal and minute volume are not separable. In addition, each limit violation triggers an alarm. This one-violation, one-alarm scheme has come under criticism because nuisance alarms cannot be distinguished from 'real' alarms. Other third-generation ventilators fare little better.

Notwithstanding minor improvements, such as setting the number of consecutive violations of high-pressure limit that trigger the high-airway-pressure alarm (introduced on the Bear 2), most manufacturers appear to have acted independently or followed someone else's lead, but without any genuine awareness of the need for a more globally integrated alarm strategy.[70] From a well reasoned but brief clinical perspective, MacIntyre and Day present a comprehensive ventilator-based alarm system.[70] Proposals such as these can be expected to stimulate manufacturers to work together toward the definition of a more efficacious ventilator-oriented alarm package.

Toward a Globally Integrated Alarm Strategy

Before I address the requirements of a globally integrated alarm strategy, some fundamental questions must be asked. Near the top of any list lies a question that asks the meaning of a limit violation. For any violation placing the patient at severe risk, the answer is unequivocal: enunciate the highest level of emergency.[71] Ventilator shutdown, loss of gas supplies, disconnection or occlusion of the patient circuit, all identify such situations. But what of other limit violations, such as the limit for low exhaled tidal volume? Most likely one single tidal volume measuring less than the low limit carries little urgency. A momentary pulse of a yellow light, including logging of the event, probably satisfies the requirement for notification and recordkeeping. If single-limit violations lack sufficient risk to the patient, what criteria must be met before trivial risks become urgent? Perhaps clinicians would agree that repetitive limit violations eventually place the patient at risk. Does this mean that any parameter, successively violated for some specific duration, must cause the annunciation of an urgent alarm (audible and visual)? If the answer is even a tentative Yes, who has the obligation to set all of the appropriate limit boundaries? Any serious attempt to pursue such reasoning eventually points to considerable uncertainty.

Fortunately, thoughtful effort has been devoted to solving some of these issues. With respect to annunciation, ISO Draft Proposal 9703.2, dated December 1, 1987,[71] offers specific audio and visual recommendations for three levels of notification: general warning, general caution, and advisory. This document takes into account the psychological aspects of annunciation and the audible sound conveying the level or urgency. Although no ventilator manufacturer has fully implemented the recommendations of ISO 9703.2, some of the newer anesthesia machines offer more comprehensive alarm strategies embracing some of the concepts embodied in this document. Ventilator manufacturers cannot afford to lag too far behind.

Any comprehensive, global alarm strategy must take into account both limit-setting and annunciation. The recommendations of ISO Draft Proposal 9703.2 appear well intentioned, but the document says little about the smart algorithms that must ascertain whether a violation rates a general warning, a general caution, or an advisory. It is tempting to suggest that, whatever the algorithmic design, limit-setting must be automatic. Built-in nomograms could provide initial boundaries subject to practitioner review, given that the patient's weight was initially identified. Once ventilation began, statistical analyses could provide updated limits based on actual data. Currently active boundary limits could be displayed on an appropriate screen, available for review and modification by the practitioner. Whatever the attraction of intelligent alarms with hierarchical annunciation, careful design, testing, and evaluation must be undertaken before such concepts gain even a modicum of credibility.

Whether, when, or under what conditions intelligent alarms become implemented, their success will directly depend on the design of comprehensive algorithms running on fast, powerful microprocessors. Rather than the current condition of one-violation, one-alarm, these algorithms will constantly monitor all patient-related parameters to determine impending patient risk. Rather than identifying a specific indicator like high-pressure limit, the ALARM violation might be accompanied by the comment, "`Violation of high-pressure limit due to sustained decrease of lung-thorax compliance`." When the practitioner recognizes the general warning, there should be no uncertainty about the level of urgency, as is the case today. Even before the general warning, the practitioner will have recognized the general caution, identifying that the condition of the patient has shifted from an earlier, acceptable state. Thus, the meaning of general warning will be unequivocal: "Come immediately, risk is imminent." Without sophisticated, nomogram-based algorithms, running under the control of powerful microprocessors, such global alarm strategies would be unthinkable.

Because of the voracious appetite of microprocessor electronics for copious quantities of data, a general monitoring umbrella could continually survey all ventilator settings—old as well as new. Such an umbrella monitor could compare all new settings to old settings and then warn the practitioner of logical inconsistencies. For example, suppose that the current setting for V_T were 600 mL, with a low V_T alarm setting of 450 mL. While conducting a routine check of the patient and ventilator, the practitioner increased the set V_T from 600 mL to 750 mL but forgot to raise the limit setting for the low-V_T alarm. An umbrella monitor would catch this error immediately. As long as the algorithm saw repetitive changes (an indication that a practitioner was addressing the ventilator), no error warning would be annunciated. When the umbrella monitor sensed no further manipulation of the ventilator (defined as an interval of x seconds without a recorded manipulation), software would issue a visual advisory. The longer the interval of inactivity, the more aggressive the advisory would become. Eventually the monitor would become suitably aggressive to demand attention. Such an algorithm has undergone limited testing, with generally positive results. Whether expanded testing would gain practitioner acceptance remains uncertain. Whatever the uncertainty of user acceptance of such an umbrella monitor, microprocessor electronics could manage the task with relative ease.

Measurement of Respiratory Mechanics

However the terms "respiratory mechanics" or "weaning parameters" are identified, microprocessor electronics possess the capacity to automate all of these types of maneuvers, measured or computed within the patient-ventilator domain (the reader is directed to Reference 72 for a detailed discussion of the monitoring of derived variables). Although many of today's microprocessor-controlled ventilators offer one or a number of algorithmically controlled maneuvers, the paucity of supportive or critical data in the literature suggests that these features receive little use. Part of the problem may stem from the very lack of supporting data. Without such data to demonstrate accuracy and efficiency, practitioners experience little incentive to change. The manufacturers also may have to bear part of the responsibility for low practitioner acceptance. Not all of their maneuvers perform to practitioner expectations; no ventilator offers a complete set of maneuvers. For example, when Puritan-Bennett designed their algorithm for negative inspiratory pressure, their engineers were unwilling to believe that the selectable occlusion interval should last from 10 to 30 seconds. Errors in the algorithms must be corrected and efficacy demonstrated before clinical acceptance can be expected to increase.

Table 2 contains a list of the respiratory mechanics weaning parameters that might be expected from an up-to-date, third-generation ventilator.[72-74]

The placement of maneuvers such as those listed above under algorithmic control endows a ventilator with unique advantages. First, variation among practitioners vanishes. The algorithm itself becomes the standard (assuming the requisite accuracy). All practitioners who learn how to initiate a maneuver can be said to demonstrate comparable accuracy. Second, the initiation of many maneuvers requires that the ventilator be changed often from its current settings. In a stressful setting such as the ICU, practitioners may forget to return the ventilator to its current settings at the conclusion of a maneuver, sometimes to the detriment of the patient. Under microprocessor control, the ventilator always reverts to the correct current settings. Third, the microprocessor has access to information unavailable to the practitioner. And further, if such information were available, the calculations required to yield a useful parameter would place it well beyond bedside relevance.

As an example, consider the simple square-waveform technique used to estimate lumped (artificial airway + patient) resistance. The clinician sets the mandatory waveform to square and selects an interval for the inspiratory pause (plateau). Next, the ventilator is commanded to deliver the square waveform with plateau. The clinician must collect three discrete values: the flow at end-inspiration, the peak airway pressure at end-inspiration, and the plateau pressure. Placing the three values in the equation,

$$R_{static} = \frac{P_{end\text{-}inspiration} - P_{plateau}}{\dot{V}_{end\text{-}inspiration}}.$$

yields an estimate of R_{static}. Errors abound in this exercise, however: the interpretation of end-inspiratory pressure, plateau pressure, and the best estimate for end-inspiratory flow (how can we know that the set flow is the true value at end-inspiration?). Notwithstanding these obvious errors, which may not be too damaging, there is another error that does not lend itself to ready dismissal. Figure 19 illustrates this error. The quantity $P_{end\text{-}inspiration} - P_{plateau}$ underestimates ΔP_R because at end-plateau, $P_{plateau}$ overestimates P_{lung}. During the plateau, some of the volume stored in the patient circuit is driven into the lung as $P_{end\text{-}inspiration}$ and P_{lung} converge to the common value, $P_{plateau}$. Although this error is generally negligible in the range of normal adult tidal volumes and compliance, it becomes relevant at smaller tidal volumes and lower compliances. Whatever the magnitude of the correction, a microprocessor, running under an appropriately designed algorithm, handles the calculations with seeming simplicity.

Processing of Data

Internal Processing

In previous sections, mention has been made of the relative ease with which microprocessor electronics manage real-time calculations and consistency checks. Given that a design for a ventilator contains an absolute pressure transducer, all gas volumes can be readily expressed and manipulated in BTPS units. Knowledge of the compliance of the patient circuit, combined with the standard measure of end-inspiratory airway pressure (P_{aw}), enables the pneumatic subsystem to deliver the set V_T to the patient's airways in the face of changing lung-thorax compliance and resistance. Any simple or

Table 2. Respiratory Mechanics Weaning Parameters Measured by Third-Generation Ventilators

Common Name	Measured Parameter	Comments
Static resistance, R_{stat}	Measured at end-inspiration, using the square waveform followed by a plateau	Valid only for a controlled breath. Patient assist most likely invalidates measurement.
Static compliance, C_{stat}	Measured after exhalation from any mandatory breath (volume- or pressure-based) ending with a valid plateau	Valid only for a controlled breath. Patient assist most likely invalidates measurement. Ideally, algorithm should provide for measurement of and correction for $PEEP_i$.
Dynamic resistance, R_{dyn}, and dynamic compliance, C_{dyn}	Simultaneously measured during any mandatory inspiration (volume- or pressure-based)	Valid only for a controlled breath. Patient assist invalidates measurement. Not invalidated by $PEEP_i$.
$P_{0.1}$ or P_{100}	Value of airway pressure measured after an inspiratory effort lasting 100 ms	Ventilator's pneumatic system must remain closed for first 100 ms or measurement is invalid. Patient must remain unaware of measurement.
NIP, P_{Imax}	Maximal value of patient's negative inspiratory pressure, measured with occluded patient circuit	Measurement requires conscious patient effort of 10 to 30 s of occluded chemoreceptor drive. No consensus whether expiratory valve is to remain open or closed.[74]
Vital capacity, V_C	Maximal, consciously generated exhaled volume	Requires patient cooperation, not a "forced" maneuver.
Intrinsic PEEP ($PEEP_i$)	Pressure in alveoli at transition from exhalation to inspiration	Requires perturbation of patient's breathing pattern. Generally measured by an expiratory pause begun at the moment of "transition." Measurement invalid if patient contracts expiratory or inspiratory muscles.
Peak inspiratory flow	Maximal value of smoothed inspiratory flow	Easy to measure, but highly influenced by selection of PSV.
Mean spontaneous inspiratory flow	Mean value of flow measured during spontaneous inspiration	Easiest to compute from V_T/t_i.
t_i/t_{tot}	Inspiratory time expressed as a weaning indicator[74]	Easily computed.
f/V_T, where f = spontaneous rate in breaths/min and V_T in L	Value proposed as a weaning indicator[74]	Easily computed.

complex corrections are readily applied to data as they are processed by algorithms and eventually become useful information.

Because microprocessor control implies discrete or discontinuous manipulation of data and events (contrasted to continuous control in an analog design), even the most irrelevant minutiae are available for capture, processing, and display. In a microprocessor design, conscious decisions must be made to limit the amount of captured data. With lax attention to detail, a system as complex as today's third-generation ventilators becomes vulnerable to the 'Christmas tree' effect—an overwhelming abundance of data and lights.

In my view, the sophistication of third-generation ventilators compares with the sophistication of the personal computer near the end of the 1970s. To reiterate a statement made earlier

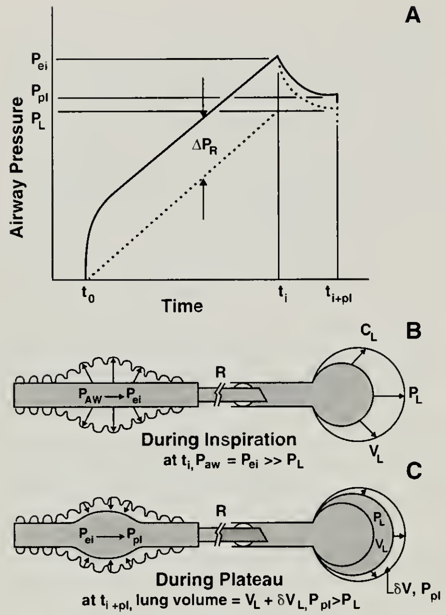

Fig. 19. A depiction of lung-ventilator interaction during a maneuver designed to measure static airways resistance (R_{static}). This maneuver yields a point measurement, ie, R_{static} represents an estimate at end-inspiration. At T_0, the ventilator begins a machine-triggered (not patient-triggered), square-flow waveform (Panel A). During delivery of the breath ($\dot{V}$ = constant), a 'constant' ΔP develops across all airways. The dashed line rising in parallel with the airway pressure trace illustrates the P-T profile in the 'lung.' The difference between lung and airway (patient circuit) pressure (ΔP_R) is proportional to R_{static}. At t_i, breath delivery ends and a plateau begins. When the plateau ends, the pressures in the lung and patient circuit equilibrate at $P_{plateau}$. Also, during breath delivery the complete patient circuit 'charges,' acquiring a finite volume increase due to $P_{end\text{-}inspiration}$ (Panel B). As the plateau proceeds, some of this accumulated volume empties into the lung, driven by $P_{end\text{-}inspiration}$ (Panel C). At end-plateau, the volume lost by the patient circuit is given by $\delta V_C = (P_{end\text{-}inspiration} - P_{plateau})\, C_C$, and for the lung, the volume gained equals $\delta V_L = (P_{pi} - P_{lung})\, C_L$. But $\delta V_C = \delta V_L$, and after rearrangement,

$$P_{lung} = P_{plateau} \frac{C_C}{C_L} + 1 - \frac{C_C}{C_L} P_{end\text{-}inspiration}.$$

This result shows that $P_{lung} \neq P_{plateau}$, as generally assumed. For the sake of interest it might be well to ask about the error propagated to the estimate of R. Knowing the true value of R,

$$R_{true} = \frac{P_{end\text{-}inspiration} - P_{lung}}{\dot{V}},$$

versus the estimated value of R

$$R_{estimated} = \frac{P_{end\text{-}inspiration} - P_{plateau}}{\dot{V}},$$

we can solve for the percent error in $R_{estimated}$:

$$\%\ \text{error} = \frac{R_{true} - R_{estimated}}{R_{true}} \times 100.$$

After substitution and rearranging,

$$\%\ \text{error} = \frac{1}{C_L/C_C + 1}.$$

As an example, assume typical values for an adult: let C_C = 2 mL/cm H_2O and C_L = 30 mL/cm H_2O:

error in R = 6%.

For a small pediatric patient attached to an adult patient circuit, assume C_C = 3 mL/cm H_2O and C_L = 12 mL/cm H_2O:

error in R = 20%.

This example illustrates that the solutions to errors too cumbersome to be taken into account during bedside calculations are easily designed into algorithms.

in this paper, the practitioner appears willing to accept sophistication veiled in the guise of simplicity. The bridge between the seemingly incompatible notions of sophistication and simplicity is software. However, before the logical and complex tasks can be relegated to software, multidisciplinary design teams must develop and validate fail-safe algorithms. My sense of the field of ventilator design suggests that this effort has begun,[75-81] but the fruits of the current efforts will not be realized for several more years. Isolated breakthroughs could appear at any time, most certainly, but a full Lotus 1-2-3 equivalent is not yet a reality.

Continuing in a similar vein, advanced microprocessor designs have the capacity to provide a much greater umbrella of logic-checking than that offered on current third-generation ventilators. As a simple example, consider the I:E logic in Puritan-Bennett's 7200. The software engineers began with the requirement that mandatory, volume-based ventilation be disallowed if combinations of peak inspiratory flow, tidal volume, flow waveform (all of which specify t_i), and respiratory rate (which specifies the breath interval, t_{tot}) cause the I:E to exceed 3:1. When this simple algorithm is embedded in the umbrella software that monitors all keyboard entries, each new value of t_i or t_{tot} gets checked for:

$$\text{New } t_i > 75\% \ t_{tot}\ ?$$

> If No, accept new selection. If Yes, reject new selection and (cryptically) suggest solution to avoid rejection, knowing which parameter invoked the rejection.

It appears reasonable to suggest that more broadly applicable, logic-checking, umbrella algorithms of the type alluded to above could succeed if the ventilator were informed of relevant patient data. Such data might include patient type (neonate, pediatric, adult), ideal body weight, description of artificial airway (size and type), and possibly disease classification. Leaving aside disease classification, identification of the other three data would enable software to set safe boundaries, ventilator settings, and emergency ventilation—and to optimize the parameters that specify the quality of flow and pressure delivered to the patient. Unknown at this time is the receptiveness of the respiratory care community to such ideas. Umbrella algorithms similar to those just identified would bear some cost. At issue, then, is the practitioner's perception of such algorithms. Ideas and their utility represent different issues. If umbrella algorithms establish credibility, they could become standard in all sophisticated ventilators of the future.

Processing Data for Distribution Outside of the Ventilator

Most third-generation, microprocessor-based ventilators (and the analog-based Siemens 900C) provide a digital-data transmission link to extra-ventilator devices, such as remote PCs and monitors. Whereas this feature has generally been well received by the respiratory-care community, the lack of continuity across the transmission protocols adopted by different manufacturers has limited installation to a few sites that have the resources to write specialized send, receive, format, and display software.[64] Using their unique and proprietary software, the Siemens 900C and 900 Servo Computer Module are able to communicate with Siemens monitors. In 1989, SpaceLabs provided an interface module that linked their PC monitor with the Puritan-Bennett 7200a, and in 1991 Hewlett Packard released a similar module that allowed their Component Monitoring System (CMS) to link with a 7200a or a 900C. Manufacturers of other monitors plan to introduce proprietary interface modules linking their devices to specific ventilators. As yet, however, no commitment exists to link all monitors with all ventilators. With continued efforts by the Federal Government and third-party payers to constrain medical costs, manufacturers will come under increasing pressure to standardize on universal communication protocols. An example is the medical information bus (MIB) proposed by IEEE.[69] While acceptance of this protocol, or for that matter, any other protocol, still suffers from a lack of universal commitment, such an expectation seems reasonable.

Data-communication protocols of the type envisioned here are well handled by microprocessor-based hardware. Once the issues of protocol design are resolved, medical devices and receiving stations (monitors) would communicate over specially designed networks. The situation would be much the same as with LANs (local area networks) in the PC environment. A working prototype of the MIB network operates continuously at LDS Hospital, Salt Lake City, Utah, under the direction of Dr Reed Gardner and his colleagues.[69] Monitors at each bedside, in central stations, and in other specific locations, can display the data from any device actively connected to the MIB. For a more in-depth understanding of a real-time, respiratory monitoring workstation, the interested reader is directed to two just-published (1992) references from the laboratory of Prakash in the Netherlands.[82,83]

Associated Issues

Systems Management

Before concluding this paper with a discussion of such ancillary ventilator features as service and automatic testing, I wish to devote some time to a highly important and sometimes emotional issue. This topic bears many seemingly incompatible names: closed-loop control, protocol medicine, decision advice, artificial intelligence. To one degree or another, all of these concepts have one aim, that of reducing some element of human decision making and replacing it with an algorithm. Because in medicine the paths from disease, to cause, to effect, to remedy, to recovery, eventually become identified—usually by applying the experimental method—the individual steps can be organized into an algorithm. Much of the natural world in which we humans find ourselves can be reduced to algorithms. This is true for baking a cake, flying an airplane, playing chess,[84] or recovering from illness.

The essence of algorithmic control is quantitative information. Algorithms define how a system or function behaves. Thus, to effect control, one must specify at least two pieces of information: the desired behavior and the current behavior. In the case of a patient-ventilator system, current information is supplied by sensors. Identification of the current state allows the control algorithm to act on the system to achieve the desired behavior. Complex systems simply require more knowledge about the current behavior, supplied by more sensors, and more complex algorithms with which to achieve the desired control.

What evokes emotion regarding algorithmic control is the degree to which human decisions are replaced by machine decisions. More than this simplistic statement underlies the issue of machine control, however. Consider, as an example, modern military aircraft. Without actually controlling the directional surfaces of these complex jet-powered fighters, military fighter pilots routinely fly these aircraft as if they were in control. Depending on one's view of the situation, the pilots do and do not control their aircraft. The pilot manipulates the control stick and the resultant forces are sensed by a computer-based, flight-control system.[85-87] The directional surfaces, then, are positioned by feedback-controlled hydraulic actuators under the command of the computer-based flight system. Who or what controls the airplane depends on the position taken by the observer. The pilot determines what he wants the plane to do by exerting pressure on the control stick, but all subsequent action is the result of a computer-based, flight-control system run by a 'smart' algorithm. Taking this example one step further, the reader will soon learn that the control of these sophisticated, complex aircraft exceeds the capabilities of the pilot. Without the computer, the plane could not be flown at its maximum level of performance.

Before leaving the example of smart aircraft, I wish to challenge the reader with a few more thoughts. The motion picture "Top Gun" may be of help here. Remember, all Navy pilots complete the standard flight-school program, which includes academic courses along with a great deal of skills training. Once in a squadron, pilots experience some additional course work but mainly increased skills training. "Top Gun" emphasized the extreme need for the perfection of those skills that could spell the difference between survival and nonsurvival. The pilots were not the least concerned about the type of control system in their aircraft. It was a given that sophisticated aircraft require sophisticated flight-control systems. The pilots also knew that without their skills, the stated mission of the aircraft would cease to exist. (Note the implied contrast here between the mission of piloted aircraft and pilotless aircraft. The Cruise missile flies entirely by computer—no pilot—but it has only one mission: fly undetected to the target site. It also does not return.) The important point regarding smart, piloted aircraft is that the pilot and the plane operate harmoniously to complete complex missions too unpredictable to be programmed in advance.

Whether there exists a parallel between the missions of fighter pilots and clinicians and between smart aircraft and smart medical devices remains to be decided at some future date. It does appear from the literature[53-58] that the topic of smart medical devices operating under the control—advisory or direct—of intelligent algorithms receives ever-growing attention. When interest runs this high, the question is no longer whether an idea will become a reality, but when and in what form.

The Upgradable Ventilator

Until relatively recently, product development followed a common theme. Each variation of a product appeared as a different model. This was true regardless of the type of device: consumer product, commercial tool, or medical device. If a customer wanted to purchase an optional configuration of a model, the order was specified on the purchase contract. In some instances a product could be returned to the manufacturer for the installation of additional features. In my experience, the annual introduction of new-model automobiles epitomizes the one-model, one-configuration concept.

Although open to speculation, it may be more than coincidence that the market leader in the United States is the Puritan-Bennett 7200 Series microprocessor ventilator,[88] an upgradable product. Several factors may bear on this issue. First, microprocessor control opened the way for the on-site addition of customer-ordered features. Second, the size of the ventilator market (approximately 4,600 pediatric-adult ICU ventilators sold in 1991 in the USA)[88] limits the economies of scale that any one manufacturer can bring to bear on the production process. By building a subsystem core—for example, pneumatic, electrical, and electronic systems, and software—one may somewhat mitigate this dilemma. Packaging the ventilator's core with different external modules allows the manufacturer to offer unique models, each perhaps configurable by specific, easily added software options. Puritan-Bennett achieved considerable success employing the concept of upgradability, and newer offerings like the Infrasonics Adult Star also appear well designed to take advantage of upgradability. In the fast-paced medical marketplace, upgradability allows a microprocessor-based product to live a longer productive life through enhancement and new features added on-site via constantly revised software.

Education and Training

Respiratory care training programs are rarely able to train their students on the most up-to-date ventilators. As federal allocations for health care diminish, fewer medical centers are able to maintain the position of education coordinator. My experience indicates that as these positions become scarcer, medical and technical directors of respiratory care will look to the ventilator manufacturers for increased training and training materials.

Given that most new-generation ventilators operate under microprocessor control, no technical impediment prevents these devices from operating in a training configuration. The monitor manufactured by SpaceLabs operates in a PC mode, allowing an operator to connect a keyboard and to write/run programs to and from an internal floppy disk.[68] If ventilators could communicate with PCs, or if they contained an internal PC function like the SpaceLabs monitor, they could operate in a training mode. Along with the release of new software enhancements and options, manufacturers could release new educational software. Depending on the size of a respiratory care department and the number of ventilators purchased from a given manufacturer, a limited number of educational stations could be equipped. In combination with the newest laser-disk technology (for example, Data Star Education Systems and Services Inc, Edmonton, Alberta, Canada), training sessions could provide outstandingly realistic learning situations.

Safety, Service, and Preventive Maintenance

Microprocessor-based designs have forever changed the way customers and manufacturers view safety, service, and preventive-maintenance functions. Each time a ventilator is turned on, sophisticated algorithms check the operational readiness of the entire electrical, electronic, and software subsystems in a few to several seconds. With the patient off the ventilator and with some operator interaction, elaborate and thorough algorithms direct, in a matter of a couple to a few minutes, one, two, or possibly three levels of pneumatic and operational verification tests that previously would have required many tens of minutes to hours to complete. Error codes or word statements immediately flag failed subsystems and, in many cases, the responsible component(s). Once ventilation begins, logic and consistency-checking constantly monitor for improper function. Error detection can cause one of two responses: a brief shutdown lasting but seconds while software attempts a restart, or—if the error falls under the critical definition—software invokes a separate backup-ventilation function. This emergency ventilation could take more than one form. For example, backup ventilation on Puritan-Bennett's current 7200 ventilator is conducted by an analog circuit separate from the microprocessor. Ventilators having two or more primary microprocessors and paired gas-delivery valves (as found in the Dräger IRISA and Infrasonics Adult Star) could invoke a form of emergency ventilation that would maintain the current settings with, at worst, a switch to either 100% air or 100% oxygen, given that the failure involved the loss of only one critical component (eg, a single flow-sensor, gas-delivery valve, pressure transducer, or microprocessor). With the FDA's (Food and Drug Administration's) current interest in software safety,[89] redundancy and operational monitoring may become more regulated and less the choice of each manufacturer.

The litigious character of American society has caused the medical profession to become acutely aware of quality control. Designing a life-support device—a ventilator, for example—around a microprocessor enables the quality-control function to acquire new dimensions. Invoking the ventilator's internal, software-directed, self-test routines markedly shortens the time required to perform operational verification procedures, while thoroughness increases. Although self-testing concepts have become more sophisticated since their introduction in the first microprocessor-controlled ventilators, considerable room for improvement remains.

The issue to be resolved, perhaps, pertains to complexity. Without calibrated internal standards (eg, a voltmeter and a calibrated pneumatic test instrument) to monitor key signals and operational performance, internal self-test routines monitor only consistency, not accuracy. The low cost of powerful personal computers may provide a solution to the problem of affordable quality assurance for ventilators. Such computer systems are widely used in many biomedical departments today.[90] Ventilator manufacturers could supply software programs for each unique product. When a given ventilator was connected to the PC via a special cable, software would read the specifications appropriate for the ventilator under test. With a pneumatic test instrument also connected to the PC, software, aided by the biomedical person, would sequentially test every facet of the ventilator. Such test programs are routinely used by manufacturers of computer-based equipment for intermediate and final test, but in this special case the manufacturer exercises total control over all facets of design and test, including training of personnel and selection of test equipment. With many institutions of differing size, complexity, and sophistication, standardization may be more difficult to achieve. The American Association for Medical Instrumention (AAMI) appears well aware of these issues, and it seems reasonable to suggest that time and cooperation between the medical community and manufacturers will elevate the quality-control function to the level necessary to ensure timely and cost-effective solutions.

Service and preventive maintenance, whether performed by factory representatives or by factory-trained biomedical personnel, bear many similarities to the quality-control function. Logically, these two activities are extensions of a quality-control program. If newer-generation, microprocessor-controlled ventilators were fitted with disk drives or their equivalent, as mentioned earlier, special service-related software might be run in the expectation of obtaining greater detail about problems discovered during normal operation. Given a sufficiently powerful software program internal to the ventilator, either resident in discrete memory chips or downloaded from the internal disk drive (possibly including an attachable keyboard, thereby turning the ventilator into a personal computer), a special modem link to a factory-service computer seems to offer little improvement over the on-site, computer-based system. What does appear reasonable is the application of more sophisticated and thorough, on-site, computer-based, quality-control procedures. Such system improvements will most likely enable manufacturers to offer longer and/or optionally configured warranty periods. The 5-year warranty introduced by Puritan-Bennett in 1990 and now offered by most other ventilator manufacturers serves, perhaps, as an example of such a system improvement.

Preventive maintenance today represents a relatively benign activity. Typically, the biomedical person checks the hour meter of a ventilator to determine whether it is time to perform the factory-specified maintenance. This determination could easily be monitored by software, yielding an hours-to-maintenance report with every initiation of an extended self-test or service requirement.

In Conclusion

I accepted the assignment to write this paper because it provided an opportunity to address the respiratory care community about a topic of importance to both clinicians and manufacturers. However, before further comment, some clarifying statements are in order. First, the ideas and observations expressed here do not necessarily represent the official position of my employer, the Puritan-Bennett Corporation. Instead, this article is the result of the myriad experiences to which I have been a party while experimenting, reading, writing, lecturing, conversing, being a member of design teams, and just plain thinking and dreaming. Second, I have attempted to present a balanced view, primarily from the positions of science and technology, clinical need, and manufacturing feasibility. The clinical community hears much from its own members but considerably less from its manufacturing partners. I have spoken about this partnership before in lectures and in talks. It is a subject about which overstatement rather than understatement is preferable. Simply stated, with the high cost of product development, manufacturers can little afford to waste their time and capital pursuing unrealistic products. Only when manufacturers and the medical communities they serve become partners will needed products be developed and sold at a fair price. And third, I have assiduously avoided comparing specific products. To the contrary, I have attempted to focus on ideas and issues rather than on solutions.

Microprocessor technology has captured the imagination of many of us, and we frequently see articles speculating about the ultimate or perfect product. With the sophistication of today's technology, reinforced by visual and perhaps personal experience, the 'do-everything' product appears evermore a reality instead of a fantasy. I touched on some of the relevant issues in this area with my discussion of closed-loop ventilation. And because I have heard the comparison of smart ventilators and smart airplanes mentioned in lectures, talks, discussions, and journals, it seemed instructive to indulge again in this comparison. There is no doubt that ventilators, like a plethora of other medical and nonmedical products, can become and are becoming smarter. The issue that begs for attention and resolution is one of operator education. By whom and how will operation occur? Modern aircraft can get smarter because the pilots believe in their aircraft and will acquire the skills necessary to operate them. No pilot ever walks out to a new aircraft with manual in hand and proceeds to fly it without hours and hours of training. Even with lavish attention to ease of operation, no new third-generation ventilator can or should be operated without some minimal level of training. Ventilators, like airplanes, do become more sophisticated with time. Someone who has marginal skills with an older-generation ventilator will most likely feel overwhelmed by the operation of a new, third-generation device. This reaction resides at the very core of our humanness.

One of the most urgent requirements expressed by the respiratory care community is the need for training. No ventilator manufacturer or medical institution has fully satisfied this deficiency. A recent demonstration of the Data Star product in Chicago as part of the 1992 meeting of the American College of Chest Physicians was most impressive. This and other similar learning concepts must play a stronger role sooner rather than later. If manufacturers teamed up with companies like Data Star and designed their ventilators to function secondarily as learning tools, training would acquire the dimensions of the flight simulator, which has been so successful for pilots. The benefits could be worth the cost.

In this article I have said little about the disadvantages that might be ascribed to microprocessor-controlled ventilators. This view most likely stems from my training (in my earlier life I was a Marine jet fighter pilot). Taking a long view, I can argue that humans exhibit an enormous capacity to exploit their environment to multiply and amplify their abilities. Humans, for better or worse, are tool makers. By harnessing the power of leverage, the inclined plane, the winch, animals, water, oil, combustion, electricity, light, tides, wind, and the like, humankind have discovered countless strategies to satisfy their needs and wants. It is not so much the microprocessor itself, which traces its origins to solid state physics, that should be focus of concern, but software. Each new technology requires taming. The FDA has correctly identified software as the issue to be understood. The solutions to bad software design focus on management, design strategy and review, controlled qualification and validation, and redundancy. The issue is not whether devices should be analog-based rather than digitally based, but how to ensure that microprocessor-based, digital designs operate as intended. A penetrating look at firms that rely on digital design and software technology for their products reveals intense involvement in programs aimed at ensuring the highest level of safety and quality.

And so, in conclusion, I hope that I have left readers with new knowledge or perhaps sharper insights than they had before reading this paper. Some of the ideas reviewed here appear in preliminary forms today. Others will require years of investigation before they come to fruition. Others will be abandoned, and some will undergo marked transformation by the time that you finally see them. Remember, what you want and need will be the result of a partnership relationship, the rules of which are only dimly perceived today. With vision and cooperation, tomorrow will bring products with new solutions along with genuine and lasting improvements.

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Computerized Ventilation Monitoring

Josef X Brunner PhD and J David Thompson RRT

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Introduction

A ventilator is one of the most complex pieces of equipment in the intensive care unit, and can have a bewildering array of control knobs and displays. It can be 'told' to deliver almost any pattern of flow and pressure at any rate and with any timing! In spite of all this technology, mechanical ventilation is often not very well understood or applied. The lack of safe and efficient closed-loop controlled ventilation is bold evidence of this contention.

Most ventilators already display many measured variables. Unfortunately, these variables are often more representative of the machine than of the patient. The major reason is that sensors for flow and pressure are located inside the machine and not close to the patient's airway. This introduces errors, especially with long tubing, humidifiers, and leaks. Another problem is that CO_2 is rarely measured and if its measurement is available it is not combined with flowsensor output. The results are often so poorly presented (or sometimes 'not presented'—ie, hidden under a switch) that their availability may not be recognized by clinicians. Alarms are still poorly handled in the state-of-the-art ventilators. Finally, none of today's ventilators makes therapy suggestions on the basis of measurements made (consultancy mode).

In this paper we address computerized lung-function measurement in intubated, mechanically ventilated patients. In particular, lung function indices, their calculation, and their potential clinical use are described. Other very important aspects of computerized monitoring—such as sensor-artifact rejection, data display, user interfacing, and smart alarms—are outside the scope of this paper.

The hypothesis is that if pertinent sensor data are combined with sound physiologic models, mechanical ventilation will become better understood and easier to manage. Breath-hold maneuvers and manual gas sampling will become obsolete through application of mathematical models, better sampling sites, and appropriate sensors. These developments will in time increase the reliability and clinical usefulness of the data while reducing the complexity of the sampling procedure.

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Physiology of Ventilation

The ultimate goal of lung ventilation is to bring O_2 into and to remove CO_2 from the cells in the tissue. This process is generally termed "gas exchange." However, such gas exchange takes place over several stages and between various kinds of tissues (including blood) and may better be called "gas exchanges." Figure 1 conceptually depicts the various stages.

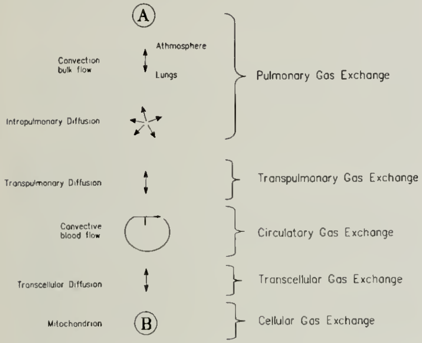

Fig. 1. Stages of gas exchange. Arrows point in both directions to indicate exchange of CO_2 and O_2. Point A denotes "atmosphere." Point B denotes "cell."

In mechanical ventilation of passive patients, gas is forced into the lungs by excess pressure (positive pressure) applied to the airway opening. As a result, intra-alveolar and intrapleural pressures rise during inspiration. (This is in contrast to spontaneous breathing during which the aforesaid pressures fall during inspiration.) Subsequent exhalation is passive and is brought about by the recoil forces of lung, diaphragm, and chest wall. The pressure-flow-volume relationship is governed by the mechanical properties of the total pulmonary system. The total compliance (C_{tot}), the total resistance to airflow (R_{tot}), and the alveolar pressure at the end of exhalation (intrinsic PEEP, or $PEEP_i$) determine the pressure-flow-volume relationship. While the positive-pressure excursions cause gas to flow in and out of the lungs, positive pressure has a negative impact on lung tissue and pulmonary hemodynamics. The clinical goal, therefore, is to minimize the pressure excursions while maintaining lung ventilation.

Adequate ventilation, however, is still no guarantee of adequate gas exchange. Unless the conducting airways are washed out by the tidal volume, gas exchange will be ineffective. It is therefore essential to know how much of the tidal volume is wasted in the conducting airways. This volume is sometimes called "series dead space" (V_{dS}).[1]

Although under certain conditions, V_{dS} correlates well with the anatomic dead space, generally, it does not. V_{dS} can be larger than the anatomic dead space if the alveoli of the first respiratory bronchioles are unperfused (that is, such alveoli are functionally, nonexistent while anatomically they are still pre-sent). Under such circumstances, V_{dS} can be said to include part of the physiologic dead space. V_{dS} can also be smaller than the anatomic dead space if emptying of the lungs is asynchronous. As a result, V_{dS} can vary grossly, even in a given patient, depending on pathology and mode of ventilation. It is therefore of value to know the V_{dS} in each patient in order to titrate appropriate tidal volumes and rates.

The gas that passes the series dead space finally enters the alveolar spaces and is thus called gross alveolar ventilation ($\dot{V}_{gA}$). Unfortunately, passing the series dead space does not guarantee adequate gas exchange either. Theoretically, all ventilated alveoli could be unperfused by capillary blood. The result would be mere alveolar dead-space ventilation ($\dot{V}_{dA}$). In normal lungs, however, only about 10% of the alveolar ventilation enters alveolar dead spaces; the rest is effective alveolar ventilation ($\dot{V}_A$). It is this $\dot{V}_A$ that actually removes the CO_2 from the lungs and, at the same time, moves O_2 into the lungs. The clinical goal, therefore, is to minimize V_{dS} and $\dot{V}_{dA}$ while maintaining $\dot{V}_A$.

At this point it is worthwhile to critically review C_{tot}, R_{tot}, $PEEP_i$, V_{dS}, $\dot{V}_{dA}$, $\dot{V}_{gA}$, and $\dot{V}_A$. These elements do not exist in reality, and anyone who asks a pathologist to show him a preparation of an alveolar dead space will remain unrewarded. There are millions of compliances, resistances, and dead spaces—just as there are millions of alveolar ducts and alveolar sacs. This reality is not easy to comprehend, much less to measure. The representation of such a complex system by a few numbers (lung function indices) is thus a necessary simplification of reality.

This "lumped parameter" approach is comparable to statistical methods whereby a population can be described by its mean and standard deviation (normal distribution assumed). In contrast to simple statistics, however, the lung function indices are "weighted averages." The results depend on the boundary conditions of measurement. For example, a hold maneuver alters measured compliance. The maneuver may not alter compliance per se, but instead will alter its apparent value. Measurement of lung function indices without a special maneuver, therefore, more closely represents the state of the patient.

Lung Function Indices

This section describes traditional and computerized methods used to measure pulmonary function in intubated patients. The prerequisite for computerized methods is the measurement of gas flow ($\dot{V}_{aw}$), airway pressure (P_{aw}), and CO_2 concentration between the Y-piece of the ventilator and the endotracheal tube.

Total Pulmonary Compliance (C_{tot})

C_{tot} is an index that describes the compliance of lung, chest wall, and diaphragm. The traditional way to assess C_{tot} is to increase P_{aw} in steps of ΔP_{aw} and to measure the associated volume (ΔV) that flows into the lungs. The ratio of volume and pressure (Equation 1) gives the value of C_{tot} at each pressure increase.

$$C_{tot}\ (\text{mL/cm H}_2\text{O}) = \Delta V/\Delta P_{aw}. \qquad [1]$$

The prerequisite is the total cessation of flow before and after each step.

Traditional Estimation of C_{tot}. Paralyzed patients on ventilators are ideal for this kind of measurement because their breath pattern can be manipulated in a number of ways. The maneuver to measure C_{tot} includes an end-inspiratory airway occlusion. This function is readily available on most modern ventilators. The task, then, is to measure end-inspiratory P_{aw} ($P_{aw\text{-}EI}$), end-expiratory P_{aw} ($P_{aw\text{-}EE}$), and the tidal volume (V_T). C_{tot} is reported as mL/cm H_2O and calculated as

$$C_{tot} = V_T/(P_{aw\text{-}EI} - P_{aw\text{-}EE}). \qquad [2]$$

The crucial steps are the accurate measurement of V_T and complete occlusion at both end-inspiratory and end-expiratory times. Most people, however, measure V_T at the expiratory outlet of the ventilator and do not apply end-expiratory occlusion. The former leads to an overestimation of V_T and C_{tot} due to additional volume compressed in the ventilator circuit during inspiration. The latter causes underesti-mation of $P_{aw\text{-}EE}$, overestimation of the difference ($P_{aw\text{-}EI} - P_{aw\text{-}EE}$), and therefore underestimation of C_{tot}.[2]

Computerized Method To Measure C_{tot}. Wald et al[3] and Uhl and Lewis[4] have described a method to automatically correct the errors mentioned. The proposed method requires the measurement of gas flow and pressure directly at the endotracheal tube. It is based on Rohrer's equation and takes into account the pressure drop over flow-resistive elements (ΔP_{flow}) and pressure buildup on compliance elements (ΔP_{volume}). This is expressed in Equations 3 and 4 and explained in Figure 2 using a pneumatic model.[5]

$$\Delta P_{aw} = \Delta P_{flow} + \Delta P_{volume}. \qquad [3]$$

$$\Delta P_{aw} = R_{tot} \cdot \dot{V}_{aw} + 1/C_{tot} \cdot V, \qquad [4]$$

with V as the integral of $\dot{V}_{aw}$ over time.

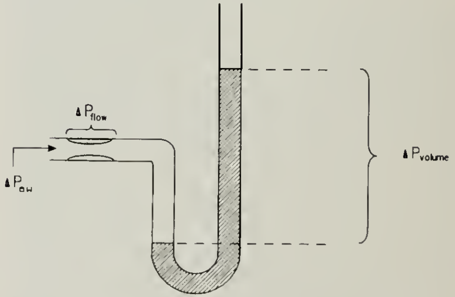

Fig. 2. Simplified schema of pressure-flow-volume relationship. An obstructed tubing is connected to a water manometer (hatched area is water). Resistance causes pressure drop (ΔP_{flow}) in presence of flow. Compliance causes 'backpressure' (ΔP_{volume}) due to volume displacement. Total pressure drop (ΔP_{aw}) is the sum of ΔP_{flow} and ΔP_{volume} (Equation 3).

Equation 3 is valid for every point in time. So is Equation 4, which is the expansion of Equation 3. Theoretically, only two sets of simultaneously measured values of P_{aw}, $\dot{V}_{aw}$, and V are needed to solve Equation 4 for R_{tot} and C_{tot}. In practice, however, the P_{aw}, $\dot{V}_{aw}$, and V signals are noisy and would render such a method imprecise. For this reason, data are collected 60 times per second, which yields a large number of equations for any given breath. The mechanics of 'distilling' C_{tot} out of this series of equations is called linear least squares fitting and was originally introduced by Gauss. Details of the method, including all formulas, can be found elsewhere.[6] Note that in Equations 3 and 4, the system is thought to restore itself to the complete equilibrium position after each breath. Exceptions to this are dealt with in detail later—in the section on intrinsic PEEP.

Bertschmann et al[7] compared the breath-hold method with the computerized technique in normal-lung patients and in patients suffering acute respiratory insufficiency. The authors also investigated the influence of $PEEP_i$ on C_{tot} measurement. They concluded that both methods yield identical results as long as the prerequisites of total end-inspiratory and end-expiratory occlusion are fulfilled. Jonson et al[2] earlier described the artifactual effects of high end-expiratory flow when end-expiratory occlusion is lacking.

Discussion. The traditional method requires end-inspiratory and end-expiratory periods of no flow. The mathematical method does not require any hold and may be superior. It is important to note that both the traditional approach and the mathematical method assume that R_{tot} and C_{tot} are constant. This assumption may be invalid in ARDS patients at very high or very low airway pressures. Under those circumstances, the mathematical method yields a mean C_{tot}, averaged over the entire pressure range, while the traditional method gives only the pressure-volume relationship at the end of inspiration. There is evidence that, because of the different techniques, the mathematical method tends to yield lower values in ARDS patients than does the traditional method.[7]

Clinical Applications. C_{tot} was measured with the mathematical method in a set of 26 patients with no pulmonary pathology after open heart surgery and was, on average, 64 ± 12.5 mL/cm H_2O. C_{tot} was slightly affected by V_T and increased with increasing V_T. This effect was statistically significant but clinically irrelevant.[6]

C_{tot} can be reliably measured only in totally passive patients with no respiratory muscle activity. In spite of this limitation, C_{tot} can be very helpful in tracking the time course of a disease and identifying abnormal conditions during mechanical ventilation. C_{tot} measurement is crucial in adjusting the pressure levels and I-E ratios during pressure-controlled-ventilation (PCV). However, too little is known clinically to give simple guidelines. On the one hand, a decreasing C_{tot} calls for high airway and high intrapulmonary pressures, which is undesirable because it may increase the incidence of barotrauma; on the other hand, an elevated C_{tot} increases the expiratory time constants, resulting in an increased mean alveolar pressure from dynamic hyperinflation ($PEEP_i$, or auto-PEEP). The tidal pressure excursions might not be so large as with the low C_{tot}, but the mean alveolar pressure may be much higher and may impair pulmonary blood flow. It is thus very difficult from a theoretical point of view to decide what C_{tot} is best for a given patient.

C_{tot} measurement is theoretically possible only in mechanically ventilated patients. There is evidence, however, that by using increased pressure-support values, one can calculate a reasonable estimate even in spontaneously breathing subjects.[8]

Total Resistance to Pulmonary Gas Flow (R_{tot})

R_{tot} determines the pressure drop (ΔP_{flow}) from the airway opening to the alveolar spaces, with respect to flow. This means that the ΔP_{flow} occurs only as long as gas is moving in or out of the lungs. When gas flow ceases, the pressure drop becomes zero (see Equation 4). This effect is the basis for the traditional estimation of R_{tot}.

Traditional Estimation of R_{tot}. Again, paralyzed patients on ventilators are ideal subjects in whom to measure R_{tot} with the old method called "the interrupter technique."[9,10] The maneuver is to occlude the endotracheal tube quickly, which must stop airway flow immediately. During passive inflation, this causes a drop in airway pressure (ΔP_{flow}). Dividing ΔP_{flow} by the $\dot{V}_{aw}$ that immediately preceded the airway occlusion will yield R_{tot} (Equation 5).

$$R_{tot} = \Delta P_{flow}/\dot{V}_{aw}. \qquad [5]$$

Alternatively, an end-inspiratory pause can be used in much the same way. This method, however, works only with inspiratory flow waveforms in which flow is not zero at end-inspiration.

Computerized Method To Measure R_{tot}. The method of Wald et al[3] and Uhl and Lewis[4] can also be used to calculate R_{tot}. In fact, the least squares fit yields both C_{tot} and R_{tot} simultaneously and with identical mathematics. Thus, solving Equation 4 for C_{tot} implies the solution for R_{tot} and vice versa.

To illustrate the method, Figure 3 shows a measured pressure-volume loop (solid curve) with superimposed fitted pressure-volume curve. As a result of that fit, R_{tot} and C_{tot} are computed (10.3 cm $H_2O \cdot s \cdot L^{-1}$ and 50 mL/cm H_2O, respectively, in the example of Fig. 3).

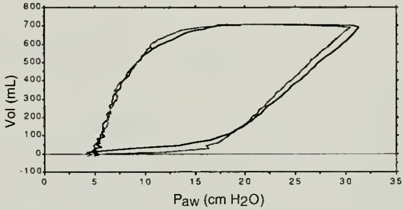

Fig. 3. Measured pressure-volume curve (solid line) and fitted curve (broken line). The latter is a result of the least squares fitting procedure. Note slight but systematic deviation on inspiratory limb of curve (right).

Discussion. The basic assumption of the method is that R_{tot} and C_{tot} remain constant throughout one breath. If this is not true, the least squares approach yields a weighted average of the R_{tot} and C_{tot} distributions.

While the fit in Figure 3 is certainly very good for clinical purposes, it deviates systematically from the measured curve during inspiration. This can be due to the fact that R_{tot} and C_{tot} are not constant during the breathing cycle—or even differ between inspiration and expiration. At least for R_{tot} in chronic obstructive lung disease patients, the latter effect has been known for years.

An important contribution to R_{tot} is the endotracheal tube. The pressure-flow relationship is described by the equation $\Delta P = a\dot{V}^b$, where a is a constant depending on the shape of the tube cross-section, the viscosity and density of the gas, and the length and diameter of the tube, and b is a dimensionless number describing the shape of the curve.[11,12] The associated resistance ($\Delta P/\dot{V}$) depends on flow and on various factors such as bending, mucus deposits, and adapters used. Table 1 contains typical values, measured over a range of 0.100 to 3 L/s.

Table 1. Pressure-Flow Relationships* of Rusch Endotracheal Tubes

Tube No.	a†	b‡	r§
6.0	24.2	1.85	0.991
6.5	16.8	1.83	0.995
7.5	8.7	1.78	0.994
8.0	7.0	1.74	0.984
8.5	5.9	1.74	0.995

*Pressure drop expressed as $a\dot{V}^b$ for flows from 100 mL/s to 3 L/s.
†a = resistance at 1 L/s.
‡b is a dimensionless number describing the shape of the curve.
§r = correlation coefficient.

The resistance of the endotracheal tube is not constant. It depends on the position in the trachea and on the presence of mucus secretions. A bent tube has a higher resistance than a straight tube, even if it is not kinked at all. Typical values for a #8 tube are given in Table 2.

Clinical Applications. R_{tot} was on average 8.2 ± 2.3 cm $H_2O \cdot s \cdot L^{-1}$ in 26 patients after open heart

Table 2. Pressure-Flow Relationships* in Unkinked #8 Endotracheal Tube at Different Angles from Straight to U-Shaped

Angle	a†	b‡	r§
180° (U-shaped)	7.265	1.674	0.997
90° (L-shaped)	6.855	1.699	0.996
0° (straight)	6.221	1.715	0.995

*Pressure drop expressed as $a\dot{V}^b$ for flows from 100 mL/s to 3 L/s.
†a = resistance at 1 L/s.
‡b is a dimensionless number describing the shape of the curve.
§r = correlation coefficient.

surgery.[6] In those patients, neither acute nor chronic lung diseases were clinically apparent. The values include the resistance of the endotracheal tube.

One interesting application of R_{tot} is the continuous monitoring of the endotracheal tube. If it becomes obstructed, R_{tot} will rise and reflect the increase in resistance.[12] Furthermore, R_{tot} can increase dramatically in acute asthma and is therefore a valuable indicator of bronchoconstriction.

In one patient, for example, R_{tot} was 25.7 cm $H_2O \cdot s \cdot L^{-1}$ prior to treatment with a bronchodilator. The arterial P_{O_2} was low (59.3 torr) and auscultation revealed inspiratory and expiratory wheezes. Tracheobronchial suctioning did not improve the R_{tot} or oxygenation. After administration of aminophylline and high intravenous dosage of prednisolone, the patient's condition improved. P_{aO_2} rose to 72 torr, and R_{tot} fell to 11.5 cm $H_2O \cdot s \cdot L^{-1}$.

Intrinsic Positive End-Expiratory Pressure ($PEEP_i$)

PEEP is widely used as a counterbalance to forces that lead to collapse of alveolar spaces and consequently to a reduction of lung volume. Modern ventilators incorporate an expiratory valve that automatically closes when the ventilator circuit pressure (P_{aw}) drops to an operator-preset level (PEEP/CPAP). The assumption is that alveolar pressure equals airway pressure at the end of expiration. If, however, expiratory airway flow does not approach zero, there will always be a difference between alveolar pressure and airway pressure (see section on R_{tot}). Under such circumstances, the $PEEP_i$ can be much higher than the external PEEP. Of course, the effect is not normally displayed on ventilators, and this fact leads to a number of names: auto-PEEP,[13] occult PEEP,[14] inadvertent PEEP,[15] and PEEP in disguise.[16]

Traditional Estimation of $PEEP_i$. $PEEP_i$ differs from externally applied PEEP because expiratory gas flow is not zero. If the airway opening is transiently occluded, gas flow will stop and $PEEP_i$ can be measured at the airway opening. This maneuver is easy in totally passive patients; in spontaneously breathing subjects, however, careful analysis of the tracheal or esophageal pressure waveform is needed.

Computerized Method To Measure $PEEP_i$. The basis for this calculation is once again Rohrer's equation of lung mechanics (Equation 4). Because the assumption of "complete equilibration at the end of each breath" must be dropped, a new term ("K") must be added to Equation 4. K has the dimensions of pressure and is introduced in the following equation:

$$P_{aw} = R_{tot} \cdot \dot{V}_{aw} + 1/C_{tot} \cdot V + K,$$

where K is a direct measure of $PEEP_i$. P_{aw}, airway flow ($\dot{V}_{aw}$), and cumulative volume (V) are all functions of time.

This means that Equation 6 is valid throughout the entire breath. If all signals are sampled very fast (eg, 60 times per second), a series of equations is obtained. These equations are solved to find R_{tot}, C_{tot}, and K by using the least squares fit approach.

The mathematical method yields values that are very close to the values obtained with the traditional breath-hold method in ARDS patients.[17] A prerequisite again is that the patient be entirely passive. In spite of the very close agreement between the two methods, there is a small systematic difference.

Discussion. Eberhard et al[17] attribute the difference between the mathematical method and the traditional breath-hold method to the fact that the traditional method introduces a maneuver, artificially prolongs the expiratory phase, and thereby leads to a systematic overestimation of $PEEP_i$. The phenomenon can be attributed to viscoelastic effects. It should be kept in mind that the difference is very small and its clinical significance is not yet proven.

One disadvantage of the traditional method is that it will not work in the presence of air leaks. Patients with chest tubes present problems in $PEEP_i$ measurement. The reason is that during a hold maneuver, the pressure drops due to the gas leaks. A possible solution might be offered by the computerized method. Because no maneuver is needed, the 'real' $PEEP_i$ could be measured. This method is currently under evaluation at our research laboratory.

Clinical Applications. Once more it must be stressed that all the above-mentioned methods require that the patient be entirely passive; otherwise, esophageal pressure measurements must be undertaken. Nevertheless, the mathematical measurement offers tremendous opportunities in patients with COPD and in pressure-controlled inverse-ratio ventilation (PCIRV). In both cases, PEEP is a dynamic effect of the ratio of inspiratory to expiratory time (I:E) and respiratory rate. In fact, the expiratory time together with the expiratory time constant largely determine the amount of dynamic hyperinflation.[7,18] The PEEP setting on the ventilator does not reflect the alveolar pressure level ($PEEP_i$). Also, PEEP effects of a dynamic nature cannot be read off the display of today's ventilators. Breath-by-breath determination of $PEEP_i$ would therefore greatly facilitate the adjustment of rate and I:E.

CO_2 Elimination ($\dot{V}_{CO_2}$)

The obvious goal of ventilation is to remove the volume of CO_2 that is produced by the metabolic functions of the body. In a steady state, CO_2 elimination is identical to CO_2 production. However, only the former can be measured at the airway opening, and any conclusions about the latter depend on assessment of steady-state conditions. This section discusses CO_2-elimination measurement and its interpretation.

Traditional Estimation of $\dot{V}_{CO_2}$. Traditionally, expired gas is collected in a Douglas bag over a period of t_{total} minutes. The mixed expired concentration (F_{ECO_2}) is measured by a mass spectrometer or capnometer and the bag is emptied into a spirometer to assess the volume (V_{total}). $\dot{V}_{CO_2}$ is then calculated as

$$\dot{V}_{CO_2} = V_{total}/t_{total} \cdot F_{ECO_2} \qquad [7]$$

The resulting $\dot{V}_{CO_2}$ is a gas volume under conditions of ambient temperature, ambient pressure, and full saturation with water vapor (ATPS). Commonly, $\dot{V}_{CO_2}$ is reported for conditions of standard temperature, standard pressure, and no water vapor (STPD). Conversion formulas are given in physics and respiratory care textbooks.[19]

The method for measuring CO_2 elimination is very reliable and has no drift, and its accuracy is unaffected by the additional compressed volume of the ventilator circuit. It is, however, cumbersome and difficult to perform in the confines of the modern ICU.

Computerized Method for Measuring $\dot{V}_{CO_2}$. At the airways, CO_2 and gas flow are not constant. Figure 4 shows a typical CO_2 curve, whereas Figure 5 shows a flow curve of a mechanically ventilated patient. The inspiratory part of the flow curve might look unusual at first because it represents the typical flow pattern during pressure-controlled ventilation (PCV). The expiratory flow curve has the form of an exponential decay curve. Note that in spontaneously breathing subjects, the flow curve may be of different shape.

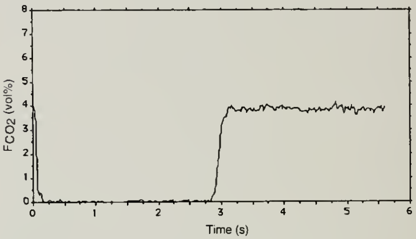

Fig. 4. Typical capnogram of a ventilated patient, showing fractional concentration of CO_2 plotted against time. Inspiration starts at Time 0, exhalation at approximately Time 2.75 s. Note that inspiratory gas contains CO_2 (rebreathing) that is washed out of the Y-piece.

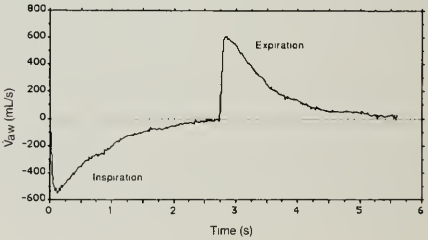

Fig. 5. Typical spirogram of a ventilated patient in pressure-controlled ventilation. Flow into patient (inspiration) is negative, whereas flow out of patient (expiration) is positive. The breath is identical to that of Figure 4.

To obtain the volume of CO_2 exhaled in one breath, the two curves of Figures 4 and 5 are combined into the CO_2-versus-volume diagram shown in Figure 6. For the sake of completeness, inspiration and expiration are plotted together in Figure 6. The area under the expiratory curve minus the area under the inspiratory curve ($\dot{V}_{ECO_2}$) is the net transfer of CO_2 out of the lungs per breath. The CO_2 elimination per minute is obtained by adding $\dot{V}_{ECO_2}$ over several breaths and dividing the sum by the total time in minutes.[20]

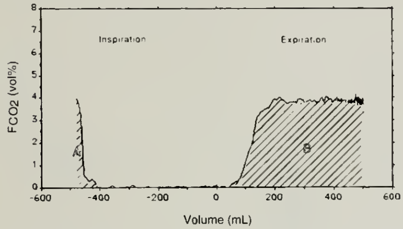

Fig. 6. Combination of capnogram and spirogram (ie, fractional concentration of CO_2 plotted against volume). Area A is inhaled volume of CO_2, and Area B is exhaled volume of CO_2. Net elimination of CO_2 is total area under Curve B – area under Curve A (rebreathed CO_2). Curve A (rebreathed CO_2). Usually, the left part of the curve (negative volumes) is omitted. The curve on the right may be divided into Phase I (no CO_2), Phase II (rapid rise of CO_2), and Phase III ('alveolar' plateau).

We have assessed the accuracy of $\dot{V}_{CO_2}$ measurement for a measuring setup consisting of a heated Jager pneumotachograph and a modified Novametrix 1260 CO_2 analyzer placed between Y-piece and endotracheal tube. A Lucite lung model with adjustable compliance and CO_2 production[6] was used as a patient substitute. CO_2 flow into the model was controlled by a calibrated Tylan FC260 mass flow controller (accurate within 1%). $\dot{V}_{CO_2}$ was varied from 150 to 550 mL/min in five steps. Variables were F_{IO_2} (0.21, 0.50, 0.90) and V_T (500, 1,000 mL). V_{ds} and C_{tot} were kept constant at 150 mL and 60 mL/cm H_2O, respectively. The result of the comparison revealed a slight overestimation of $\dot{V}_{CO_2}$ (5%) at a precision of ± 11 mL/min. The results are shown graphically in Figure 7.

Discussion. The accuracy of the computerized method of $\dot{V}_{CO_2}$ depends on the accuracy of the pneumotachograph and the accuracy of the CO_2 analyzer, and the quality of synchronization between spirogram and capnogram.[6,21] In the worst case, the errors of each of the factors add up. For example, if the relative errors for spirometry and capnometry are both ± 5%, the worst case error becomes 10%. Additionally, synchronization of spirogram and capnogram is important; small deviations of even a few milliseconds will yield gross errors (> 20%).[22]

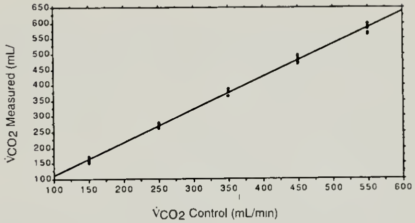

Fig. 7. CO_2 elimination measured with a pneumotachograph and a CO_2 analyzer (y-axis) plotted against calibrated inflow of CO_2 (mass flow controller, x-axis). Variables are F_{IO_2} (0.21, 0.50, 0.90) and V_T (500 and 1000 mL). Solid line is regression line ($y = 1.046x + 8.4$, $r^2 = 0.995$). Lung-model characteristics were compliance 60 mL/cm H_2O, resistanc, 5 cm $H_2O \cdot s \cdot L^{-1}$, and series dead space 150 mL.

The fact that $\dot{V}_{CO_2}$ was slightly overestimated in the above series of measurements was due to incomplete delay-time compensation. Because all signals were sampled at 60 Hz (60 measuring points per second), the smallest time increment was 16.6 ms. Consequently, the synchronization between spirogram and capnogram was limited to an accuracy of ± 8.3 ms, which causes errors in $\dot{V}_{CO_2}$ measurement.

Clinical Application. In 26 patients after open heart surgery, the mean (SD) $\dot{V}_{CO_2}$ was 194 (35) mL/min STPD.[6] Very few data are available in the literature concerning $\dot{V}_{CO_2}$ measurement in intubated patients. Much of the available literature on the subject is contradictory and the results vary greatly.[23] Most publications are restricted to the communication of end-tidal CO_2 or mixed expired CO_2 concentration. The clinical usefulness of $\dot{V}_{CO_2}$ measurement is therefore still not well defined. Potentially, $\dot{V}_{CO_2}$ is an indicator of the level of sedation, psychoemotional stress, hyperalimentation, catabolic states such as septicemia, and acute changes in pulmonary blood flow. Recently, $\dot{V}_{CO_2}$ was found to be a promising early indicator of hyperthermia[24] and therefore malignant hyperthermia. The authors concluded that $\dot{V}_{CO_2}$ is superior to the conventional indicators—end-tidal CO_2 (during spontaneous breathing), rectal temperature, mean blood pressure, and heart rate.

Series Dead Space Ventilation (V_{dS})

Series dead space is an effective in-vivo measure for the volume wasted in the conducting airways. Its determination depends on the availability of a tracer gas that indicates the transition from purely gas-conducting spaces to the gas-exchanging spaces of the lungs. Early investigators used nitrogen as an indicator,[25] which requires a well-defined and abrupt change in inspiratory gas composition. A much simpler method is to use the body's own tracer gas—CO_2.[26] The CO_2 diagram may be divided into Phase I (no CO_2 present), Phase II (rapid rise in CO_2), and Phase III (alveolar plateau).

Traditional Estimation of V_{dS}. The classic way to measure series dead space was described by Fowler.[25] His method is applicable without modification to CO_2 although it was originally defined for N_2. Figure 8 shows graphically the various steps:

1. Plot CO_2 against expired volume.
2. Identify an alveolar plateau.
3. Draw a straight line through alveolar plateau.
4. Choose a vertical line such that areas A and B become equal.
5. The intercept of the vertical line with the x-axis gives V_{dS}.

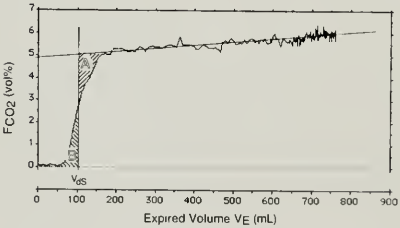

Fig. 8. Dead-space measurement by Fowler method. Alveolar plateau is defined by eye. Areas A and B must be matched by moving the vertical line left or right. Final position indicates V_{dS}.

Computerized Method To Measure V_{dS}. The basis for V_{dS} measurement is, again, the CO_2-versus-expired-volume diagram. The method is described in detail in Reference 27 and is illustrated in Figure 9. The first step is to identify Phase II of the diagram because the remainder of the analysis is restricted to Phase II. In the second step, the series dead space is calculated according to Equation 8.

$$V_{dS} = V_E^* - [V^*CO_2/F^*CO_2], \qquad [8]$$

with V_E^*, V^*CO_2, and F^*CO_2 as defined in Figure 9.

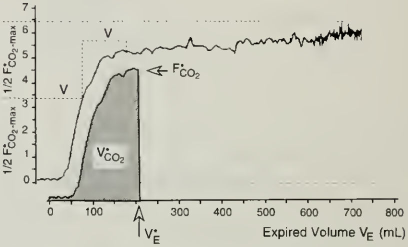

Fig. 9. Dead-space measurement according to Reference 6. The method requires two steps, focusing on Phase II and analysis of Phase II (rapid upswing of curve). Step 1: Take the maximum of F_{CO_2}, divide it by 2, and determine the concomitant volume (V). Include the curve up to V_E^* = 2 V (grey shaded) for Step 2. Step 2: Analyze the curve as cumulative distribution curve (s-shaped probability curve). See text for details.

We assessed the accuracy of V_{dS} measurement for a measuring setup consisting of a heated Jager pneumotachograph and a modifed Novametrix CO_2 analyzer placed between the Y-piece and the endotracheal tube. The same Lucite lung model was used as described earlier.[6] The model incorporates a variable V_{dS} by means of tubing added to the model outlet. Tubing volume was measured with water (± 2 mL accuracy). Gas volumes were corrected for end-inspiratory compression. V_{dS} was varied from 70 to 350 mL in five steps. Variables were F_{IO_2} (0.21, 0.50, 0.90), V_T (500, 1000 mL), and C_{tot} (30, 60 mL/cm H_2O). $\dot{V}_{CO_2}$ was kept constant at 250 mL/min. The results of this comparison are plotted in Figure 10. The mean difference was –5 ± 11 mL.

Discussion. In the comparison in Figure 10, V_{dS} was slightly underestimated (–5 ± 11 mL). This again is probably due to inaccurate delay-time compensation (see Reference 28 and $\dot{V}_{CO_2}$ discussion). A more accurate compensation would be possible by either increasing the sample rate (> 100 Hz) or interpolating between sample points. The error of less than 5% for the present setup, however, is considered clinically negligible, and higher accuracy is therefore not necessary.

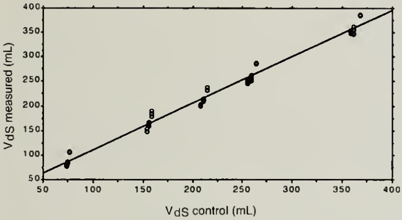

Fig. 10. Comparison of V_{ds} measurement (y-axis) with tubing volume of model lung (V_{ds} control).

The morphology of the lungs is the physiologic basis for the proposed method[29] by which the lungs can be described as a tree-like branched system. The origin (trunk) is the trachea, the terminal units (leaves) are the acini. The pathways from the trachea to the acini are of different length. Consequently, the airways associated with each pathway contain different volumes (dead-space fractions). The total of these volumes is the total series dead space.

Unfortunately, we cannot measure the total series dead space because the individual dead-space fractions empty asynchronously. To make things even worse, the contribution of each dead-space fraction is flow- and concentration-weighted.[27,29] Based on the above considerations, the CO_2-versus-volume diagram can be interpreted as a cumulative distribution function of acinar (alveolar) gas. The proposed method measures the mean value of this distribution.

V_{ds} depends on the slope of the alveolar plateau. Although this dependence is slight in most patients, it becomes significant in COPD.[30] An attempt to account for a steep alveolar plateau was successful in that it produced a V_{ds} that was close to the expected value. Yet, the clinical significance of such a mathematical maneuver is unclear because it removes information contained in the "pathological" CO_2 diagram. Therefore it may be necessary to use both methods, the original V_{ds} and the plateau-corrected V_{ds}, for maximal information.

Clinical Application. The mean (SD) V_{ds} was 167 (50) mL in 26 patients after open-heart surgery.[6] It was significantly increased with V_T, PEEP, and inspiratory flow. End-inspiratory pause reduced V_{ds}.

It is generally accepted that with conventional mechanical ventilation tidal volumes need to be larger than the anatomic dead space in order to ventilate effectively. Because anatomic-dead-space data are not readily available for each individual patient, a recommended estimate is 1 mL per pound of ideal body weight.[31] Therefore, it seems logical to base the tidal volume on body weight in order to compensate for anatomic-dead-space ventilation. However, what counts for effective gas exchange is not the anatomic dead space but its functional counterpart, V_{ds}. And V_{ds} is not very much dependent on body weight but much more so on functional variables such as breathing pattern, passive or active inflation, and even pulmonary hemodynamics. To illustrate this, Figure 11 shows data measured in patients after open heart surgery during the weaning from mechanical ventilation with IMV.[32] It is clear from Figure 11 that body weight is probably a secondary determinant of V_{ds} in adults, a fact also described by other authors.[33]

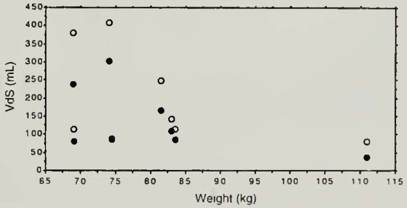

Fig. 11. Series dead space (V_{ds}) plotted against patient weight during period of weaning from IMV in 8 patients. Two points are plotted for each patient, one representing V_{ds} of mandatory breaths (open circles), the other representing V_{ds} of spontaneous breaths (closed circles). Data are from Reference 32. There is no positive correlation with weight, either with mandatory breaths or with spontaneous breaths.

Clinically, these facts may become important when tidal volumes need to be set. It does seem reasonable to base the target V_T on the actual V_{ds}.

Consequently, V_{ds} might also help to evaluate the spontaneous breathing efforts during weaning from the ventilator (ie, if V_T exceeds V_{ds} significantly, spontaneous breathing may be considered effective). Although this idea might seem plausible, there is as yet no scientific evidence to prove its superiority over the conventional approaches.

Alveolar Efficiency of CO_2 Elimination ($AlvEffCO_2$)

The series dead space is one of the causes of inefficient gas exchange. Another cause is the alveolar dead space—ie, the functional equivalent of spatial and/or temporal $\dot{V}_A/\dot{Q}$ mismatch. The basis for $\dot{V}_A/\dot{Q}$ assessment is the $\dot{V}_A/\dot{Q}$ diagram[34] (Fig. 12). Mixed-venous (v), arterial (a), and inspiratory (I) P_{CO_2}/P_{O_2} points are the cornerstones of the diagram. The bold lines are the lines of constant RQ for blood (top, nearly horizontal) and gas (from Point I to i). The lines intercept on the $\dot{V}_A/\dot{Q}$ at the 'ideal' point (i). Point i gives the P_{CO_2}/P_{O_2} of an ideal lung with complete and undisturbed gas exchange. This point is the basis for all shunt and V_D/V_T equations and requires the measurement of mixed-venous and arterial blood gases.

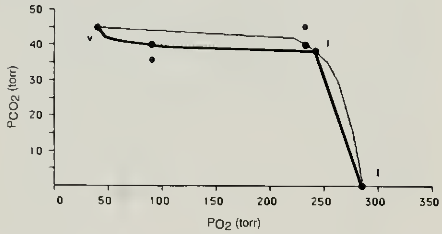

Fig. 12. Classical $\dot{V}_A/\dot{Q}$ diagram. Bold lines are RQ lines (constant RQ) for blood (v-a-i) and gas (I-i). They intersect at the ideal point (i). Thin line is $\dot{V}_A/\dot{Q}$ line. $\dot{V}_A/\dot{Q}$ is zero at mixed-venous point (v) and approaches infinity at inspiratory point (I). Dashed horizontal line represents usual method to calculate ideal P_{CO_2} and intersects with $\dot{V}_A/\dot{Q}$ line at the effective point (e). Modified from Reference 35 for F_{IO_2} of 0.40.

In clinical practice, the measurement of mixed-venous blood gases requires the presence of a pulmonary artery catheter. For this reason, the 'trick' of using arterial P_{CO_2} as the 'ideal' P_{CO_2} has become generally accepted; however, the error introduced by this trick is shown graphically in Figure 12. The 'effective' point denotes the P_{CO_2} on the V/Q line that is identical to the arterial P_{CO_2}. Because the blood RQ-line is so flat, the error remains small. If, however, the arterial point is shifted toward the mixed-venous point, the error increases. This will happen with large $\dot{Q}_s/\dot{Q}_t$ (venous admixture).

Traditional Estimation of V_D/V_T. Traditional estimation assumes that arterial P_{CO_2} represents the ideal P_{CO_2}. Expired gas is collected in a Douglas bag and the mixed expired CO_2 is measured with a mass spectrometer or capnometer. Arterial blood is sampled simultaneously and analyzed for P_{CO_2}. The latter is corrected to body temperature and converted to fractional concentration of dry gas. Finally, Equation 9 is used to calculate V_D/V_T.

The assumption is that if the lungs were ideal gas-exchanging spaces, all expiratory gas would have a concentration of CO_2 that equaled the ideal F_{CO_2} (arterial). Therefore, the difference between mixed-expired and ideal CO_2 concentration is attributed to dead-space ventilation.

$$V_D/V_T = 1 - (F_{ECO_2}/P_{aCO_2})\,(P_B - P_{H_2O}), \qquad [9]$$

where P_B = barometric pressure and P_{H_2O} = water vapor pressure, and P_{aCO_2} = CO_2 partial pressure corrected to body temperature.[36] P_{H_2O} is 47 torr at 37°C.

Problems with the measurement in patients on ventilators are associated with the collection of expired gas. Because the ventilator cannot be disconnected, gas is sampled at the expiratory outlet. Compressed gas in the tubing dilutes the expiratory gas and thus the F_{ECO_2} is decreased artifactually. This leads to an overestimation of V_D/V_T. The error increases with increases in respiratory rate and end-inspiratory airway pressure. These errors in volume measurements are greatly reduced by collecting the expired gas through a Desautels IMV expiratory-volume manifold.[37]

Computerized Method To Measure $AlvEffCO_2$. This method takes the V_{ds} into account. Consequently, and if the lungs are ideal, the 'mixed alveolar' concentration of CO_2 (F_{ACO_2}, Equation 10) equals the ideal alveolar CO_2 concentration. Any alveolar dead spaces introduce a difference between F_{ACO_2} and ideal alveolar CO_2 concentration.[38]

$$F_{ACO_2} = \dot{V}_{CO_2}/(V_T - V_{dS}). \qquad [10]$$

The ratio between mixed alveolar and ideal alveolar CO_2 concentration was originally introduced in 1957 by Severinghaus and Stupfel[38] and is termed alveolar efficiency of CO_2 elimination.[6,39] It is abbreviated as AlvEffCO_2 and expressed in % (100% means ideal).

$$AlvEffCO_2 = (F_{ACO_2}/P_{aCO_2})\,(P_B - P_{H_2O})\,(100). \qquad [11]$$

Like the other computerized methods, this one also requires the measurement of airway flow and CO_2 concentration directly at the airway opening.

Discussion. Admittedly, the information content of V_D/V_T and AlvEffCO_2 is different. The former contains V_{dS}, whereas the latter does not. This fact, however, is rarely realized, and V_D/V_T is thus usually taken as a measure of alveolar dead-space ventilation. A plot of V_D/V_T against AlvEffCO_2 (n = 42) reveals the poor correlation between the two ($r^2 = 0.102$) (Fig. 13, data from Reference 6).

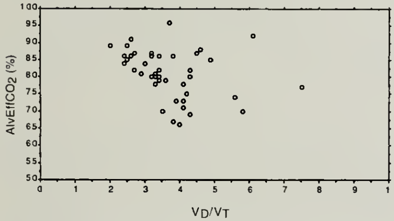

Fig. 13. V_D/V_T plotted against AlvEffCO_2 (n = 42). Data taken from Reference 6. Correlation is poor ($r^2 = 0.102$).

The validity of AlvEffCO_2 (and V_D/V_T) calculation is crucially dependent on the 'ideal' P_{CO_2}. For $\dot{Q}_s/\dot{Q}_t$ up to 20%, the arterial P_{CO_2} is a good estimate of the ideal P_{CO_2}.[35] For $\dot{Q}_s/\dot{Q}_t$ above 20%, the correct ideal point must be determined in the $\dot{V}_A/Q$ diagram. If this is not done, alveolar dead space is overestimated.

Clinical Applications. In 26 patients after open heart surgery, mean (SD) AlvEffCO_2 was 81 (7) %. AlvEffCO_2 was significantly reduced with large V_T and high inspiratory flow. An end-inspiratory pause increased AlvEffCO_2 slightly. No dependence on PEEP was measured.[6]

Although introduced in principle many years ago,[38] AlvEffCO_2 and V_{dS} are not commonly used in management of ventilated patients. Lack of suitable equipment might be one of the reasons. A lack of appreciation of the clinical benefits, based on disappointing experience with V_D/V_T, might be another reason. Yet, AlvEffCO_2 used in conjunction with V_{dS} may help to elucidate complex pathophysiologic phenomena. To illustrate this potential, data from a patient with acute bronchoconstriction, before and after treatment with aminophylline and high-dose intravenous prednisolone, are plotted in Figure 14.

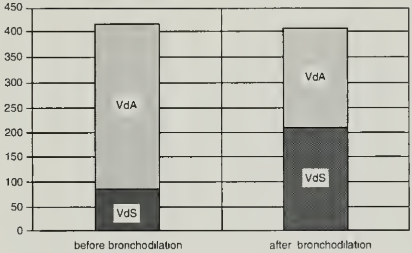

Fig. 14. Respiratory dead spaces during acute bronchoconstriction (left) and after treatment (right). Physiologic dead space (V_D) is the sum of V_{dS} and V_{dA}. V_D remains constant, whereas V_{dS} increases and V_{dA} decreases after treatment.

For compatiblity reasons, AlvEffCO_2 is presented as alveolar dead space (V_{dA}). The total of V_{dS} and V_{dA} is physiologic dead space (V_D), as expressed in Equation 13.

$$V_{dA} = (V_T - V_{dS})\,(100 - AlvEffCO_2)/100. \qquad [12]$$

$$V_D/V_T = (V_{dS} + V_{dA})/V_T. \qquad [13]$$

Figure 14 illustrates the effect of the bronchoactive drug in this particular case. During the asthma attack, R_{tot} was 26 cm $H_2O \cdot s \cdot L^{-1}$ and V_{dS} was 83 mL. When the bronchodilator was administered, R_{tot} fell to 12 cm $H_2O \cdot s \cdot L^{-1}$ and V_{dS} rose to 208 mL, both reflecting the increase in airway diameter. At the same time, V_{dA} decreased due to improved $\dot{V}_A/\dot{Q}$ matching. The fact that physiologic dead space (V_D) (or V_D/V_T, as V_T was constant throughout the measurements) remained unaltered shows the weakness of the V_D/V_T concept. In contrast, V_{ds} and $AlvEffCO_2$ plausibly document the effects of the acute pathology and their reversal.

The Future in Ventilation Monitoring

Airway Monitoring

Most ventilators measure pressures and gas flows inside the ventilator only and not at the patient's airways. This can lead to gross errors for today's modes of ventilation, especially at high airway pressure (generating an increase in compressed volume) and high gas flows (increasing pressure drop over tubing). For some respiratory modes (continuous flow), measurements inside the ventilator must even be deemed useless. In contrast, monitoring at the airways gives the real picture. Gas flow in and out of the patient is measured—not gas flow in and out of the ventilator. All the described lung-function variables are therefore based on measurements at the airways.

The greatest pressure drop normally occurs as the gas travels through the endotracheal tube (ETT) or tracheostomy tube (TT). This is the smallest diameter in the patient circuit; thus, it offers the greatest resistance to gas flow. During active inspiratory flow, the pressure measured in the ventilator circuit always overestimates the lung pressure. Conversely, during active expiratory flow, the pressure measured in the ventilator circuit always underestimates the lung pressure.

During the early development of high-frequency ventilation modes, endotracheal tubes and unique adapters were developed that provide a pressure-measuring port in the trachea. These devices are now commonly available to permit the clinician to measure intratracheal pressures directly. Measuring pressures directly in the trachea greatly reduces the error induced by the ETT/TT resistance.

In the future, airway monitoring will be the standard. It will still be necessary to monitor the ventilator's performance; however, patient monitoring is critical for appropriate therapy.

The presentation of the monitored data is critical to the clinician's ability to decipher the information. In dynamic modes of ventilation, presentation of patient flow, pressure, and volume is optimally accomplished by monitors presenting graphics of real-time plots and X-Y plots. This type of monitor is capable of providing quantitative as well as qualitative presentations of compliance, resistance, work of breathing, and auto-PEEP. As with all new technologies, these monitors do come with a price tag in both cost and handling.

Computerized Interpretation and Consulting

Data from lung-function analysis in intubated patients on ventilators have no meaning per se. The data need to be interpreted in the context of the disease and the short-term therapeutic goals. Furthermore, the interpretation must be followed by appropriate actions. The loop is closed when the lung function data are used again to verify the success of the actions.

One of the crucial steps in the above closed-loop control system is the interpretation of data in the context of the disease and the therapy.[40] This notion needs to be extended to the availability of resources, to house rules, and to local standards of care. Consequently, a computerized system for the interpretation of lung function data in intubated patients needs to be adaptable to local circumstances. This is a difficult task and requires the involvement of the users in the design of such systems. It even requires the users to maintain the system themselves in order to accommodate the changes in house rules or resources over time.[41]

It is widely believed that artificial intelligence technology will solve such problems.[40,42] However, experienced researchers in the field question the efficiency of so-called heuristic or rule-based programming. One of the most prominent opponents of this type of artificial intellligence even states that "Heuristic techniques do not yield systems that one can trust."[43] In the presence of the legal issues related to medical decision support systems,[44] this contention must be carefully considered by both vendors and users.

In contrast to heuristic programming, a structured approach is based on physiologic and/or therapeutic models. This requires more than just agreeing on stated rules, it requires the consensus of all the clinicans involved.

Furthermore, sophisticated, automated lung function analysis is needed to provide a continuously adapted database for the control algorithms.[45] We believe that the combination of consensus-based control algorithms and automatic lung function analysis will finally lead to clinically beneficial closed-loop control ventilation.

ACKNOWLEDGMENTS

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Abnormal Chest Radiograph after Central Line Placement

Debra L Medin BS RRT and Frederick P Ognibene MD

A 32-year-old Caucasian woman with metastatic breast carcinoma was admitted to the Medical Intensive Care Unit of the Warren G Magnuson Clinical Center, National Institutes of Health, for chemotherapy. This woman had undergone a right modified-radical mastectomy for breast carcinoma in March 1989 and was now being treated for metastatic disease with a combination chemotherapy protocol consisting of taxol and adriamycin. Initial physical examination revealed pain in the right lower back and leg, sternal pain, and left-side rib-cage pain. The admission chest radiograph is shown in Figure 1. For the purpose of administering the oncolytic agents, a 7-Fr, 16-cm triple-lumen catheter was introduced into the superior vena cava via the right internal jugular vein. Local anesthesia was accomplished by instillation of 3 mL of 1% lidocaine hydrochloride before catheter insertion. Physical findings were unremarkable and unchanged after the procedure. The follow-up chest radiograph is shown in Figure 2. A third chest film was obtained approximately 1 hour later (Fig. 3).

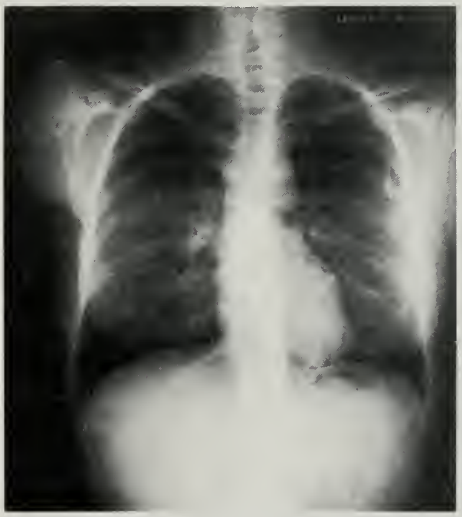

Fig. 1. Admission posteroanterior chest film of patient who had undergone right modified-radical mastectomy.

Questions and Answers
on Next Page

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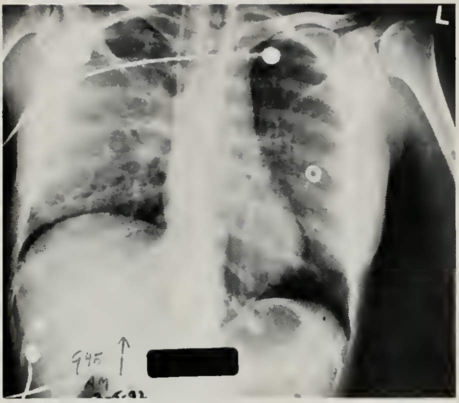

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Fig. 2. Anteroposterior chest radiograph taken immediately after internal jugular catheterization for central venous access.

Questions

Radiographic Finding: What abnormality is present in the follow-up chest film (Fig. 2) that is not seen in the admission film (Fig. 1)?

Diagnosis: What is the etiology of the radiologic finding in Figure 2?

Diagnostic Confirmation: Does the third film (Fig. 3) support this diagnosis?

Corrective Action: What corrective intervention was required to produce the change seen in the third film?

Answers

Radiographic Finding: The follow-up chest radiograph (Fig. 2) shows marked elevation of the right hemidiaphragm. The admission film gives evidence of metastatic disease in the increased nodular appearance of the lungs in the periphery. The extent of disease appears greatest in the lingular region.

Diagnosis: Hemidiaphragmatic elevation in this patient is most likely the result of phrenic nerve paralysis induced by local anesthesia with lidocaine hydrochloride.

Diagnostic Confirmation: The third film (Fig. 3) shows resolution of the elevation of the right diaphragm and, therefore, confirms the presence of an inadvertently induced, transient phrenic nerve paralysis as suggested by Figure 2.

Corrective Action: In this case, the short-acting nature of lidocaine made clinical intervention unnecessary, and paralysis resolved spontaneously.

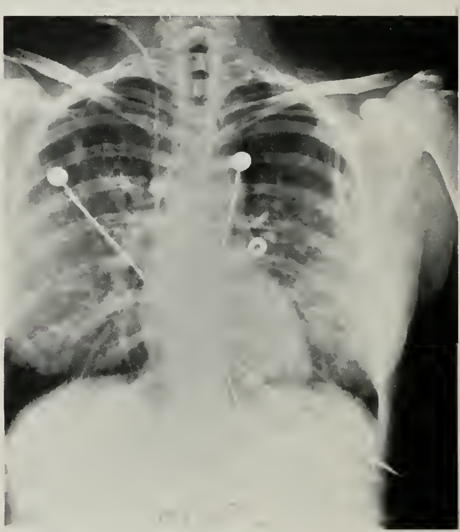

Fig. 3. Anteroposterior chest film taken about 1 hour after film shown in Figure 2.

Discussion

Cannulation of the internal jugular vein for central venous access is a relatively low-risk procedure. Indeed, the internal jugular is the vessel of choice due to the low incidence of pneumothoraces compared with other routes of access. However, it is not without other complications as presented here.

Because of the right phrenic nerve's proximity to the internal jugular vein, it is anatomically vulnerable to inadvertent puncture or anesthetic paralysis. Internal jugular cannulation requires that a needle be inserted deep into the neck just at the level at which the phrenic nerve traverses the anterior scalene muscle.[1] Through transsection of the internal jugular vein and because the phrenic nerve is just posterior and lateral to the vein at this point, trauma to or anesthetization of the nerve can occur. Iatrogenic phrenic nerve paralysis, such as that seen in this patient, has been previously documented.[2-7] Vest et al[2] and Hadeed and Brown[4] reported damage to the phrenic nerve that occurred after repeated attempts at cannulation. External compression of the nerve by a hematoma was the cause of temporary paresis described by Depierraz et al.[5] Other reports have been published associating phrenic nerve paralysis with subclavian venous catheterization and brachial plexus anesthesia.

Still other conditions exist that can cause either unilateral or bilateral diaphragmatic paralysis. These include mediastinal or bronchogenic masses producing a compressive neuropathy, poliomyelitis or pachymeningitis infections, pulmonary infarction, lead poisoning, cervical spondylitis, or any surgical procedure involving the neck or mediastinum.[8] In this last category, hematomas, which may cause phrenic nerve compression, are the most common complication associated with internal jugular cannulation.[9] Additionally, the frequency of such complications increases with repeated attempts at cannulation.[2,4] The diagnosis of diaphragmatic paralysis is confirmed by fluoroscopy. Using this radiographic technique, the lack of movement or paradoxical movement of the diaphragm is readily seen.

The morbidity of unilateral hemidiaphragmatic paralysis is minimal. Symptoms include dyspnea and tachypnea (with or without hypoxemia) and shoulder neuralgia. The physical exam reveals decreased or absent breath sounds on the affected side, immobility of the affected hemidiaphragm, and a thoracic breathing pattern. Occasionally, the paralyzed side may demonstrate a paradoxical rise during inspiration.[8] In addition to the elevation of the hemidiaphragm, atelectasis may be seen on the affected side on the chest radiograph.

The outlook in our case as for most other patients with iatrogenically induced hemiparesis is excellent. The duration of blockade of lidocaine in centrally located peripheral nerves is approximately 120 minutes.[10] Thus, recovery is usually complete in 1 to 3 hours. However, for those patients in whom trauma to the nerve has occurred, a long, slow recovery is expected, and those patients with complete transsection of the nerve will never regain function of their diaphragms. Compression paralysis requires that the source of compression be removed. Recovery depends on the resolution of the compression.

Despite their low incidence following phrenic nerve paralysis, diaphragm hemiparesis, dyspnea, and hypoxemia can occur. These pulmonary compromises may be poorly tolerated in those with cardiorespiratory disease. It is essential that venous catheterization procedures be monitored closely and that there be careful follow-up, including a chest radiograph, to ensure patient safety.

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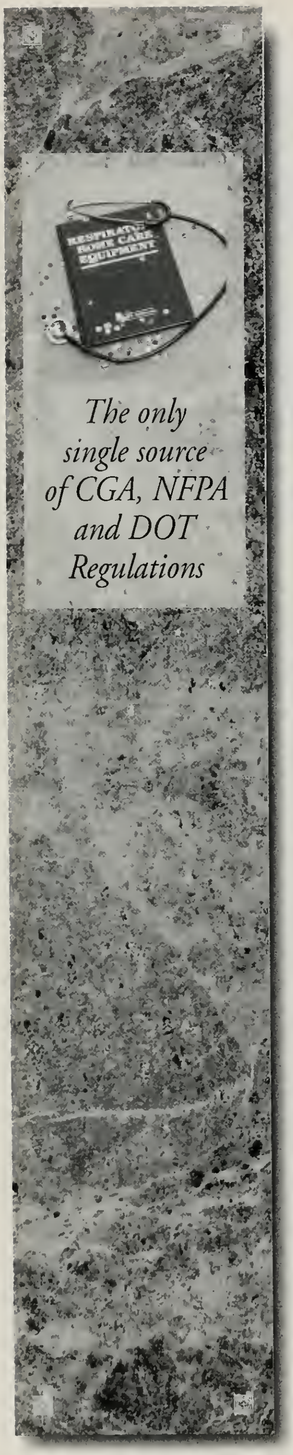

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PFT Corner #48—
An Uncommon Variant of an Upper-Airway Disorder

Jack Wanger RPFT RRT and William R Beam MD

A 39-year-old woman who had never smoked was referred to our outpatient clinic with the chief complaint of disabling chronic cough. She gave no previous history of pulmonary or sinus disease and denied symptoms of seasonal or perennial allergies. Additionally, she denied symptoms of gastroesophageal reflux and was using no medications. She denied constitutional complaints. Her symptoms of paroxysmal, nonproductive cough began after she returned to work following a leave of absence to undergo a hysterectomy. She was employed as a quality control inspector at a factory involved in the manufacture of plastic computer parts. She had been symptomatic for 4 weeks when her family physician made a diagnosis of "bronchitis" and prescribed an inhaled beta agonist and later, an inhaled corticosteroid. These measures, in addition to an 8-week leave from work, were unsuccessful in controlling her cough. Although she denied dyspnea related to cold air or exercise, she did report hoarseness and associated the cough with exposure to chemicals used at the factory. Prior to referral to our clinic, she had been terminated from her job.

During the evaluation, she was noted to have an intermittent, dry cough during conversation. Her physical examination was noteworthy for the absence of nasopharyngeal pathology and the presence of clear vesicular breath sounds without wheezes, rhonchi, or crackles. Cardiac examination results and her chest radiograph were normal. Pulmonary function studies were performed and the results are shown in Table 1 and Figure 1.

Table 1. Results of Pulmonary Function Tests before and after Bronchodilator (Albuterol) on 39-Year Old Woman

Test	Before	After
TLC (L)	5.66 (110)*	5.43
TGV (L)	2.75 (97)	2.43
RV (L)	2.45 (145)	2.40
FVC (L)	3.24 (83)	3.04
FEV_1 (L)	2.62 (80)	2.59
FEV_1/FVC (7)	81	85
FEF_{50}/FIF_{50} (%)	1.35	2.00
R_{aw} (cm $H_2O \cdot s \cdot L^{-1}$)	2.44	1.16
sG_{aw} (L/cm $H_2O \cdot s \cdot L^{-1}$)	0.23	0.26
D_{LCO} (mL · min · torr)	26.55 (95)	–
D_{LCO}/V_A	5.95 (106)	–

*Values in parentheses are percent of predicted.

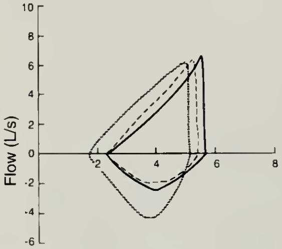

Fig. 1. The flow-volume relationships for a 39-year-old woman with asthma symptoms. The hatched line is the reference (predicted) relationship. The solid line is the flow-volume curve prior to an inhaled albuterol treatment. The dashed line is the flow-volume curve after albuterol.

Questions

1. How do you interpret the pulmonary function test results?
2. What other pulmonary function tests are indicated?

Answers and Discussion

1. Interpretation of PFT: The pulmonary function tests show mild overdistention (increased RV), normal airflow, normal diffusing capacity, and no significant response to bronchodilator. The reduced inspiratory flows and increased FEF_{50}/FIF_{50} are consistent with variable extrathoracic obstruction.

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2. Additional Tests: Although the patient denied episodes of wheezing or dyspnea and demonstrated no bronchodilator response on her tests (Table 1), a bronchial provocation (challenge) test is indicated. Chronic cough is most commonly due to one of four disorders: postnasal drip, asthma, chronic bronchitis, or gastroesophageal reflux.[1,2] History, physical examination, and methacholine inhalational challenge were diagnostic in 86% of patients in one large series of patients with chronic cough.[1] Additionally, chronic cough has been reported as the only symptom of asthma in some patients with normal spirometry but airways hyperresponsive to methacholine.[3]

A fiberoptic evaluation of the patient's nasopharynx and large airways may be indicated in light of the flattening of the inspiratory portion of the flow-volume loop (Fig. 1). Although the diagnostic yield of fiberoptic bronchoscopy in patients with chronic cough and a normal chest radiograph is low,[1,2] the flow-volume characteristics suggest the presence of a variable extrathoracic obstruction.

Results of Additional Tests

A bronchial provocation test with methacholine was performed. The results (Table 2 & Fig. 2) show no clinically important airways hyperresponsiveness, with PC_{20} FEV_1 = 14.4). (A PC_{20} $FEV_1 > 8$ suggests normal responsiveness.) Sinus radiographs and 24-hour esophageal pH monitoring revealed no evidence of sinusitis or gastroesophageal reflux. A fiberoptic bronchoscopy revealed a normal trachea, normal airway anatomy, and no endobronchial lesions or ulceration. The carina was biopsied during the bronchoscopy and results were normal. However, an additional interesting finding was noted. Prior to the endobronchial inspection, the vocal cords were examined. Although both cords appeared to move without paralysis, the patient demonstrated notable vocal cord adduction with inspiration and nearly complete closure of the vocal cords with expiration.

Table 2. Results of Bronchial Provocation Test Using Inhaled Methacholine in a 39-Year-Old Woman*

Dose (mg/mL)	FEV_1	% Change†
Baseline	2.77	–
Diluent	2.68	–
0.31	2.70	+1
0.62	2.67	0
1.25	2.70	+1
5.00	2.38	−11
10.00	2.23	−17
25.00	2.01	−33

*The PC_{20} FEV_1 is 14.4 mg/mL. See Figure 2.

†% change from diluent.

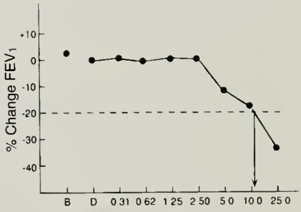

Fig. 2. Graph of results of the methacholine provocation test. The % change in FEV_1 is calculated from the FEV_1 measured after inhalation of the saline diluent (D). The dosing begins at 0.31 mg/mL and increases in doubling increments up to 25 mg/mL. A greater than 20% fall in FEV_1 was observed after the 25 mg/mL dose. The PC_{20} FEV_1 is 14.4 mg/mL.

Discussion

In the current literature, vocal cord dysfunction is not usually listed as a cause of chronic cough,[1,2] and the symptom of cough is not commonly listed in descriptions of vocal cord dysfunction.[4-6] Our patient's condition may represent an uncommon variant of an uncommon syndrome.[6-8] Patients with vocal cord dysfunction[4] have had their condition reported as Munchausen's stridor,[9] factitious asthma,[10] and emotional laryngeal wheezing.[6] The clinical features attributed to vocal cord dysfunction differ in some series but can be described as inspiratory or expiratory vocal cord dysfunction, or both.[5]

Inspiratory vocal cord dysfunction is usually characterized by inspiratory wheezing resulting from inappropriate adduction of the vocal cords. The flow-volume loop is abnormal with evidence of variable extrathoracic obstruction (ie, flat inspiratory portion and increased FEF_{50}/FIF_{50}).[8]

Expiratory vocal cord dysfunction (vocal cords are adducted during expiration, but glottis widens during inspiration) is usually characterized by expiratory wheezing. During symptomatic episodes, respiration often occurs close to residual volume and spirometry results are variable.

Inspiratory and expiratory vocal cord dysfunction may have both inspiratory and expiratory wheezes, which are commonly loudest over the larynx. Laryngoscopy when performed during wheezing shows almost complete adduction of the vocal cords throughout the respiratory cycle. Spirometry results are misleading and generally are variable, with poor reproducibility. In fact, the pronounced variability in spirometric tests may be a useful clue to the diagnosis. The flow-volume loop commonly shows variable extrathoracic obstruction but also additional expiratory limitation.[5]

Patients with vocal cord dysfunction are usually women between ages 20 and 40 years, often have psychosocial dysfunction, may have some medical background, and tend to be educated above the high school level.[6]

The definitive test for vocal cord dysfunction is fiberoptic laryngoscopy, ideally during an exacerbation. Bronchial provocation tests such as methacholine challenge and exercise may induce the paradoxic vocal cord movement. To avoid false-positive findings, the laryngoscopist needs to be aware that the vocal cords tend to close near the maximum inspiratory level (total lung capacity). Additionally, the cords may adduct on expiration with obstructive disease and some other lung diseases.[6]

In many cases, acute attacks can be relieved with a helium-oxygen mixture (70% helium and 30% oxygen). However, long-term therapeutic intervention often requires a multidisciplinary approach involving physicians, psychiatrists, and speech therapists.

Our patient's vocal cord dysfunction and cough improved after a long course of speech therapy during which she was taught to focus her attention away from the larynx and inspiration, to relax the oropharyngeal muscles, and to concentrate on active expiration.[8] It remains possible that relatively low levels of chemical irritants played a role in triggering our patient's symptoms, although environmental analysis showed chemical levels in the factory air to be within OSHA standards.

In Conclusion

In conclusion, the association between chronic cough and vocal cord dysfunction is rarely reported. Although our patient's cough could possibly have been triggered by chemical exposures during work, it appears more likely that the cough was related to her vocal cord dysfunction. While vocal cord dysfunction is an uncommon cause of chronic cough, the abnormal flow-volume loop combined with a normal methacholine challenge suggests the diagnosis. Our case represents an exception to the reportedly limited usefulness of fiberoptic bronchoscopy in a patient with chronic cough and a normal chest radiograph.

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Inspiratory Flow Patterns: The 'Shape' of Ventilation

Joseph L Rau Jr PhD RRT

Historical Perspective

The clinical importance of altering inspiratory flow patterns remains somewhat cloudy to most practitioners. Part of the reason for this may be that it is only the newer and, specifically, the third-generation ventilators that have allowed clinicians to choose from among a variety of flow patterns. An additional reason may be that the literature on the subject has been sporadic, extending over a period of years, beginning in 1947 with Cournand et al's work on pressure curves.[1] However, there has been a surprising amount of literature in that time reporting the results of research on inspiratory flow patterns. Cournand's work established that higher mean airway pressures ($\bar{P}_{aw}$) result in lowered cardiac output. Accordingly, pressure curves, which can be varied by changing the flow pattern, can influence cardiac output by increasing or decreasing $\bar{P}_{aw}$. In a series of studies, Bergman[2-4] found that higher airway pressures decrease (ie, improve) the alveolar-to-arterial-oxygen pressure gradient [$P_{(A-a)O_2}$], but concluded that the shape of the pressure waveforms was not important. In other words, for both Cournand and Bergman, it was not the shape of the pressure curve but the mean pressure that was important. In fact, the ratio of inspiratory to expiratory time (I:E) was considered of more importance in determining $\bar{P}_{aw}$ than the shape of the pressure curve.[5]

Table 1 summarizes studies of inspiratory flow patterns.[6-18] Despite differences in methodologies and subject conditions, there are common threads in most of the work. (1) $\bar{P}_{aw}$ is higher with descending patterns and lower with ascending ones. (2) P_{peak} is lower with descending and higher with ascending patterns. (3) Gas distribution is improved with descending patterns, as evidenced by lower P_{etCO_2} values. (4) A descending pattern can improve arterial oxygenation, either by improved gas distribution, higher $\bar{P}_{aw}$, or other mechanisms not yet elucidated. The work done in our laboratory suggests that the lung condition influences the airway pressures generated with different inspiratory flow patterns.

Inspiratory Flow Patterns of Third-Generation Ventilators

Terminology

The studies summarized in Table 1 supply evidence of the variation in terms used to describe inspiratory flow patterns. I use the terminology proposed by Chatburn[19] to describe operator-selectable waveforms available for machine, or mandatory, breaths of specific ventilators. (Inspiratory flow patterns of spontaneous or demand-flow breaths are not addressed.) Machine breaths are those delivered in what has been traditionally termed control, or assist-control mode, and more currently referred to as continuous mandatory ventilation (CMV) or volume-control mode, on some ventilators. Typical tracings of possible waveforms are shown in Figure 1.

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Table 1. Studies of Inspiratory Flow Patterns and Their Effects on Pressures and Other Variables

		Observations				
Investigator	Model & Flow Generator	$\bar{P}_{aw}$	P_{peak}	Gas Distribution	P_{aO_2}	Other
Lyager (1968)[6]	Lung model; Engstrom Bird Mark 8, Bennett PR-2	—	—	Poorest with ascending	—	—
Adams et al (1970)[7]	Canine model; experimental flow generator	Highest with sine & descending; lower with ascending & square	—	—	—	—
Jansson & Jonson (1972)[8]	Computer simulation	Higher with descending, lower with ascending & square	Lower with descending, higher with ascending & square	Most uniform with descending	—	—
Johansson & Löfstrom (1975)[9]	Human subjects; Servo 900	Ascending	Lowest to highest: descending, square, ascending	—	—	—
Baker et al (1977)[10]	Canine model; experimental flow generator	Highest to lowest: descending, square, sine, ascending	—	—	Most favorable with descending	V_D/V_T, C, P_{aCO_2}, C.O. most favorable with descending
Damann (1978)[11]	Human subjects; Servo 900B	Sine wave	—	Sine better than square	—	—
Modell & Cheney (1979)[12]	Canine lung injury; Emerson 3PV		—	—	10% increase in O_2 with descending vs ascending sine wave	—
Baker et al (1982)[13,14]	Canine pulmonary edema & emphysema; experimental flow generator	Highest to lowest: descending, square,	—	—	—	Lowest P_{aCO_2} with descending
Banner et al (1986, abstract)[15]	Lung model of uneven ventilation; Hamilton Veolar (4 waveforms)	—	Lowest with descending, highest with ascending	Improved with descending	—	Lowest P_{aCO_2} with descending decrease in P_{etCO_2} with descending and increasing inspiratory time
Branson & Hurst (1987, abstract)[16]	Human subjects; Hamilton Veolar (4 waveforms)	Highest for descending & square	Lowest to highest: sine, square, descending, ascending	—	—	—
Smith & Venus (1988)[17]	Pigs with high R_{aw}; Hamilton Veolar (4 waveforms)	Higher with ascending than with square, sine, or descending	—	—	—	No differences in gas exchange or hemodynamic values
Rau & Shelledy (1991)[18]	Lung model (VT-1); Hamilton Veolar (7 waveforms) baseline & low compliance	Highest with full descending, lowest with full ascending	Lowest to highest: descending, modified sine, sine, partial descending, square, partial ascending, & full ascending	—	—	—

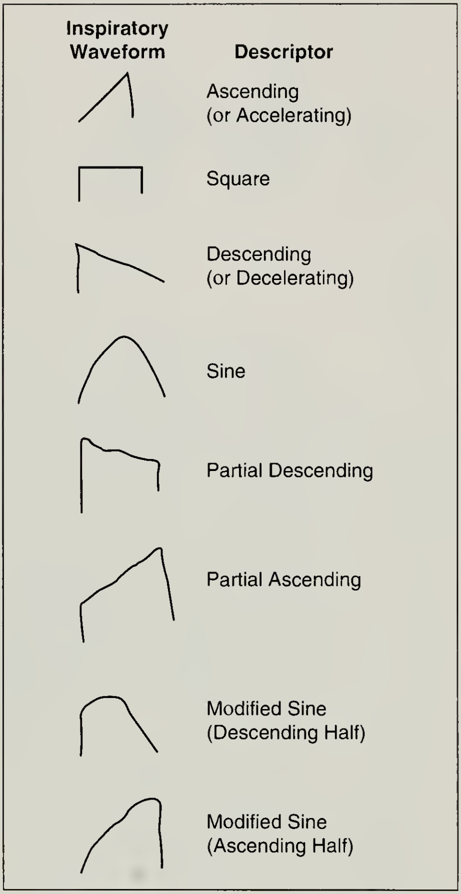
Inspiratory Waveform
Descriptor
Ascending (or Accelerating)
Square
Descending (or Decelerating)
Sine
Partial Descending
Partial Ascending
Modified Sine (Descending Half)
Modified Sine (Ascending Half)

Fig. 1. Tracings typical of the various inspiratory waveforms available with mechanical ventilators.

Rectangular: *pattern generated by a constant flow throughout inspiration; also referred to as a uniform flowrate or a square wave.*

Descending: *pattern generated by flow that begins at peak and decreases in a linear manner until the end of inspiration; also referred to as a decelerating waveform.*

Ascending: *pattern generated by flow that begins with a low value and increases until the end of inspiration; also referred to as an accelerating waveform.*

Sine: *pattern generated by flow that increases to a peak and then decreases.*

For each of the ventilators presented, the choices of inspiratory flow patterns, the primary cycling mechanism, and the controls that set minute ventilation in what is traditionally termed assist-control are listed in Table 2. These variables are provided because they can influence the effect of changes in flow patterns on the I:E and inspiratory time. The flowrate range for each machine is also provided in Table 2.

Infrasonics Adult Star

The VENTILATOR SETTINGS screen is accessed through a SCREEN CHANGE button, and ventilator setting changes are selected using a rotary SELECT knob to move the screen cursor to the function desired. By depressing the ENTER button, values are highlighted for that function (parameter) and the value is selected by using the SELECT rotary knob. Depressing the ENTER button a second time completes the selection. The available inspiratory flow patterns are shown in Figure 1 and can be described as

Rectangular—delivers a constant flow at the preset peak flow setting.

Descending—flow begins at the preset peak flow value and decreases linearly to 30% of the peak flow.

Ascending—flow begins at 30% of the preset peak flow value and increases linearly to the peak flow value.

Sine—flow begins at 30% of the preset peak flow value, increases to peak flow, and then decreases back to 30% of the peak flow, describing the waveform of a sine function.

Bear 2 and Bear 3

The inspiratory waveform is controlled by the toggle switch on the front panel. The two waveforms are

Table 2. Inspiratory Flow Pattern Capabilities in Assist-Control Mode, Primary Cycling Mechanism, and Controls Determining Minute Ventilation in Selected Ventilators

Waveform	Adult Star	Bear 2, 3	Bear 5	Bird 8400ST	Puritan-Bennett 7200	Hamilton Veolar	Hamilton Amadeus	Sechrist 2200B	Servo 900C	Servo 300
Rectangular	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Descending	Yes	Yes	Yes	Yes	Yes	Yes	Default	Yes	—	—
Ascending	Yes	—	Yes	—	—	Yes	Option	Yes	—	—
Sine	Yes	—	Yes	—	Yes	Yes	Option	Yes	Yes	—
Partial Descending	—	—	—	—	—	Yes	Option	—	—	—
Partial Ascending	—	—	—	—	—	Yes	Option	—	—	—
Modified Sine	—	—	—	—	—	Yes	Option	—	—	—
Flowrates L/min	10-120	10-120	5-150	10-120	10-120	3-180	3-180	2-120	< 150	< 180
Cycling	Volume	Volume	Volume	Volume	Volume	Time	Time	Time	Time	Time
Controls	Volume Flow Rate	Volume Flow Rate	Volume Flow Rate	Volume Flow Rate	Volume Flow Rate	Volume Rate I-Time %	Volume Rate I-Time %	Volume Rate I-Time % seconds	Volume Rate I-Time %	Volume Rate I-Time %

Rectangular—a constant inspiratory flow is delivered during inspiration at the preset peak flow.

Descending—flow begins at the preset peak flow value and decreases as pressure in the patient circuit increases. Thus, the form is somewhat variable because it depends on the level of back pressure. At 120 cm H_2O, the peak flow is reduced to 50% of its preset value.

Bear 5

The four possible inspiratory flow patterns are selected by pressing the waveform illustration on the membrane touch pad.

Rectangular—constant flow is delivered at the peak flow setting.

Descending—flow begins at the preset peak flow value and decreases linearly to 50% of the peak flow.

Ascending—flow begins at 50% of the peak flow setting and increases linearly to the preset peak flow.

Sine—flow is delivered from zero to the peak flow setting and then back to zero in a form approximating a sine function.

Bird 8400ST

Inspiratory flow pattern is selected by use of a front-panel knob, that also selects the mode to be used. Two waveforms are available in both assist-control and SIMV modes.

Rectangular—flow is delivered at a constant rate throughout inspiration at the selected peak flow setting.

Descending—flow begins at the peak flow setting and then decreases linearly to one half of the peak flow setting.

Puritan-Bennett 7200

Three inspiratory flow patterns are available for selection from illustrated waveforms on the membrane touch pad.

Rectangular—flow is delivered at a constant value during inspiration and is determined by the operator-selected peak flow value.

Descending—flow begins at the preset peak flow value and decreases to an end-inspiratory value of 5 L/min.

Sine—flow begins at 5 L/min, increases to the preset peak flow value, and then decreases back to 5 L/min.

Hamilton Veolar

The Hamilton Veolar offers the greatest number of operator-selectable inspiratory waveforms among the current generation of ventilators, with seven patterns from which to choose. Inspiratory flow patterns are accessed by use of a front-panel control labeled with illustrations of the waveforms. In the time-cycled Veolar, inspiratory time remains unchanged when the inspiratory waveform is changed.

Rectangular—flow is constant during inspiration and is determined by the tidal volume (V_T) selected and the inspiratory time (t_i) that results from the interaction between the selected rate (f) and I:E.

Ascending—flow begins at baseline (zero) and for comparable settings for V_T, I:E, and f increases to twice the rectangular wave value, to deliver the V_T in the same t_i.

Descending—for comparable V_T, I:E, and f, flow begins at twice the rectangular wave value and decreases to zero.

Sine—for comparable V_T, I:E, and f, flow begins at zero, increases to 157% of the rectangular wave value, and then decreases back to zero, in a sinusoidal pattern when graphed against time.

50% descending—for comparable V_T, I:E, and f, flow begins at 133% of the rectangular wave value and decreases to 66% at end-inspiration.

Modified sine—for comparable V_T, I:E, and f, flow begins at zero, increases to 133% of the rectangular wave value at the first third of inspiration and then decreases back to zero.

50% ascending—for comparable V_T, I:E, and f, flow begins at 66% of the rectangular wave value and increases to 133% at the end of inspiration.

Hamilton Amadeus

The Hamilton Amadeus is a simplified version of the Veolar. The usual inspiratory flow pattern is the rectangular waveform, although a second pattern is available by use of an Options (DIP) switch on the rear of the machine. The second flow pattern is the choice of the purchaser and can be any one of the remaining six waveforms described for the Veolar. The ventilator is configured for this pattern by the manufacturer prior to delivery. If the purchaser does not specify the second flow pattern, the 50% descending pattern is the standard default. When Option Switch 9 is in the ON position, the rectangular pattern is changed to the second, or alternate, pattern chosen by the user. The rectangular pattern and the other six flow patterns available are the same as described for the Veolar.

Sechrist Model 2200B

The Sechrist Model 2200B adult ventilator is a recent microprocessor-controlled, volume-preset, time-cycled entry into the ventilator market. This machine offers a choice of four inspiratory flow waveforms, accessed through a SETUP key using Routine 3. A numbered list of waveforms is then displayed in a 40-character message window and selected through entry of the appropriate number. As with most microprocessor-controlled units, the selection is completed by depressing the ENTER key.

Rectangular—flow is constant throughout the inspiratory phase, and determined by the operator-set V_T and t_i.

Sine—for comparable V_T, I:E, and f, flow increases from zero to 1.4 times the rectangular flowrate value and then decreases back to zero.

Ascending—for comparable V_T, I:E, and f, flow begins at 0 L/min, increases to 1.33 times the rectangular flowrate value for the first 50% of the inspiratory time, and then maintains that flowrate for the remaining pre-set inspiratory time.

Descending—for comparable V_T, I:E, and f, flow begins at 1.33 times the rectangular flowrate value and maintains that value for 50% of the inspiratory time; then the flow decreases to 0 L/min in the last 50% of the inspiratory time.

Siemens Servo 900C

The Servo 900C, as well as the Servo 900B, offers two inspiratory flow patterns, which can be accessed by means of the toggle switch on the front panel.

Rectangular—flow is delivered at a constant rate during inspiration and is determined by the V_T to be delivered (which is derived from the minute volume divided by the rate setting) and the t_i percentage set.

Sine—the sine is an approximate sine pattern, in which flow goes from baseline to 1.5 times the rectangular wave value (for comparable V_T, I:E, and f) and then back to baseline.

Siemens Servo 300

The Servo 300 has no operator-selectable panel control to allow choice of an inspiratory flow pattern. A preselected V_T is delivered during an operator-set t_i. The primary controls for a machine breath in assist-control, or VOLUME CONTROL to use the Servo label, are: V_T, f, t_i%, and pause time %. Flow is normally delivered at a constant rate, with the flowrate value determined by the V_T and t_i. For example, a volume of 0.8 L delivered in 1 second requires a flow of 0.8 L/s, or 48 L/min. However, the Servo 300 allows a certain modification or 'shaping' of the rectangular wave pattern. The first modification is by use of a panel control labeled the INSPIRATORY RISE TIME %. With this control, the operator can slow the rate of flow increase to the peak value of the rectangular waveform. The control is graduated from 0 to 10, indicating the percentage of the breath cycle time needed to reach the peak flow. For example, with an f of 12 breaths/min, the breath cycle time is 5 seconds. Setting the INSPIRATORY RISE TIME at 10% creates a 0.5-second period during which flow increases from 0 to a peak value. Flow then remains constant until the V_T is delivered. The peak flow value adjusts to maintain the preset inspiratory time. In effect, a pattern is created that shows an upward ramp and then levels off to a rectangular form. INSPIRATORY RISE TIME can also be used in SIMV, pressure support, and pressure control modes.

A second modification to the inspiratory flow can occur in response to patient demand, with volume-control mode. If a patient's inspiratory effort lowers the monitored airway pressure below the set PEEP level, the ventilator is put on a demand-flow basis and delivers whatever flow is needed (within the limits of the machine, up to 180 L/min) to maintain the set PEEP level. There are several 'stopping rules' to terminate this "pressure-regulated" flow, which can be found in the Operator's Manual.[20]

Considerations in Choosing an Inspiratory Flow Pattern

The mechanical properties of the lungs in relation to the mechanical movement of gas into the lung are the basis for the pressures generated during inspiration. This includes both P_{peak} and P_{aw}. During inspiration, pressure is generated by flow meeting the resistance of the airway and the gas volume overcoming the elastic recoil of the lung. A more precise statement is seen in the following equation:

$$\text{Peak pressure} = (\text{Flow})(R_{aw}) + (V_T)(\text{Elastance}),$$

where Raw is airway resistance in cm $H_2O \cdot s \cdot L^{-1}$, and elastance is pressure/volume, usually in units of L/cm H_2O.

The shape of the inspiratory flow pattern as it delivers the V_T determines the shape of the pressure curve and, especially, the peak pressure generated. This can be predicted by the clinician. For example, in an ascending flow pattern, the highest flow is at the end of inspiration; therefore, the highest pressure due to flow and resistance occurs then. At the same time, the volume delivered is largest at the end of inspiration; so, the highest pressure due to volume and elastance is at the end of inspiration. Because with an ascending pattern the maximum pressures due to flow and volume coincide in the inspiratory cycle, the ascending pattern generates the highest peak pressure compared to the other flow patterns. However $\bar{P}_{aw}$ tends to be less because most of the pressure generated occurs at only the one point in the cycle. This has been verified in the studies reviewed at the beginning of this paper. In contrast, a descending flow pattern generates the most pressure due to flow at the beginning of inspiration when pressure from elastance and volume is low, whereas pressure from volume and elastance increases toward the end of inspiration. Because these pressures are generated at opposite ends of the inspiratory phase, peak pressure tends to be lower, but mean, or overall, pressure during inspiration is more constant and higher.

For other flow patterns, such as the sine, square, and modifications to the ascending and descending patterns, peak pressure increases and mean pressure decreases as the highest point of flow in the pattern moves from the beginning to the end of the inspiratory cycle.

With high airway resistance situations, such as in severe asthma or tracheal stenosis or other obstructions, peak pressures can be reduced by avoiding any pattern with a high peak flowrate. In a time-cycled ventilator with constant inspiratory time, a partial descending or a rectangular flow pattern give lower peak pressures than a full descending pattern. With a constant inspiratory time, the full descending form requires a higher peak flow at the beginning of the breath to deliver the V_T. For example, using the Hamilton Veolar, a 1-L volume in 1 second gives the following peak flow values for the waveforms indicated.

rectangular	60 L/min
50% descending	80 L/min
sine	94 L/min
full descending	120 L/min

It is interesting to note that the descending flow pattern is similar to the variable flow pattern during pressure-control ventilation and even pressure support. The resulting pressure curve is also similar to the constant pressure seen with these modes. This constant pressure may be at least partially responsible for the beneficial effects of improved gas distribution and better P_{aO_2} values seen in some studies with a descending flow pattern.[8,10,12,15]

A final consideration in choosing inspiratory flow patterns is the effect of flow pattern change on I:E. In time-cycled machines, such as the Hamilton Veolar or the Sechrist 2200B, changing from a rectangular, or constant, flow to a varying flow does not change the I:E, which is fixed. If the flow is lower than constant rectangular flow at some point in the inspiratory phase, then the flow must be higher at some other point, to deliver the volume in the same time limit. For example, if the rectangular flow is 60 L/min, a full descending pattern must be 120 L/min at the beginning of inspiration because the flow decreases to 0 at the end of inspiration. Otherwise the volume cannot be delivered in the set time. By the same reasoning, a volume-cycled machine does not fix the I:E and the t_i. The peak flow is set by the operator as a primary ventilatory control as indicated in Table 2. If the peak flow value does not change from the rectangular pattern value when the flow pattern is changed, t_i lengthens as the volume is delivered. This concept is illustrated in Figure 2 for a full descending flow pattern, from a time-cycled and volume-cycled machine. Peak pressure may decrease more with a volume-cycled machine when changing from a rectangular to a descending pattern, than with a time-cycled unit because t_i is now longer. Critical patients may not benefit from a longer t_i and lower peak flows when work of breathing is considered. Clinicians should monitor I:E with flow pattern changes on volume-cycled machines, and if necessary, increase the PEAK FLOW setting to maintain a suitably short inspiratory time.

In Summary

Most patients requiring uncomplicated or short-term ventilatory support probably can be adequately ventilated using a rectangular or sine wave flow pattern.

Hypoxemic patients or those with low lung compliance may benefit from a descending flow pattern, with lower P_{peak} and higher $\bar{P}_{aw}$.

Patients with very high airway resistance may have lower P_{peak} with a partial descending or a rectangular flow pattern, assuming the I:E remains fixed.

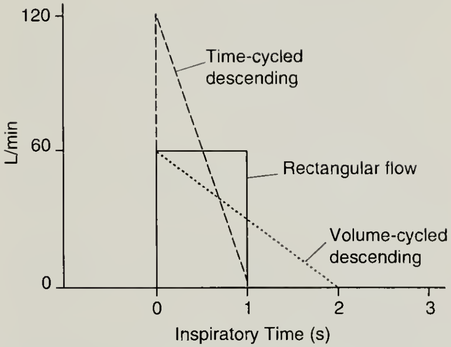

Fig. 2. As an inspiratory flow pattern is changed from a rectangular to a descending waveform, inspiratory time and I:E will vary for a volume-cycled ventilator unless the peak flow setting is manually changed. A full descending flow pattern (peak to zero flow) is illustrated. See text for discussion.

Clinicians should monitor I:E when changing inspiratory flow patterns on volume-cycled machines, to keep t_i at a suitable value.

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Retrospectroscope—1975

Pulmonary Diffusing Capacity for Carbon Monoxide ($D_{L_{CO}}$)

The brief inhalation of low, nontoxic concentrations of carbon monoxide is now a standard procedure in pulmonary function laboratories to measure the rate of diffusion of gas across the alveolar-capillary membranes of ventilated, perfused alveoli, to estimate pulmonary capillary blood volume, and to provide an index of the area and thickness of the alveolar-capillary membranes and of the number of capillaries participating in gas exchange. Some now believe that a low $D_{L_{CO}}$ may be a very early sign of pulmonary emphysema; if so, the CO test may be used widely for early diagnosis of this disease.

Who first thought of using a toxic gas for the safe measurement of an important function of the lung —and why? How did the clinical test originate? The story is divided into two parts, separated by a period of 35 years. Part one began in the 1890's when Christian Bohr, the distinguished Danish physiologist, believed that oxygen passed from alveolar gas to pulmonary capillary blood by active secretion as well as by passive diffusion.[1] And J. S. Haldane, Oxford's great respiratory physiologist, was equally convinced that lungs secreted oxygen. In 1897, Haldane and Smith[2] wrote:

> In the animals investigated the normal oxygen tension in the arterial blood is always higher than in the alveolar air, and in some animals even much higher than in the inspired air. The absorption of oxygen by the lungs thus cannot be explained by diffusion alone.

August Krogh disagreed, even though Krogh had been a student of Bohr's and was Bohr's assistant from 1899 till 1908. In a series of experiments that required devising the Krogh spirometer, the Krogh microtonometer for measuring the tension of gases in arterial blood, and the Krogh bicycle ergometer, August and his wife, Marie Krogh, systematically disproved the theory of oxygen secretion. A. Krogh's 1910 experiments led him to the unqualified conclusion that "The absorption of oxygen and the elimination of carbon dioxide in the lungs takes place by diffusion and diffusion alone. There is no trustworthy evidence of any regulation of this process on the part of the organism."[3] But Marie Krogh still believed it necessary to prove that diffusion alone was also sufficient to account for the *maximal* transfer of oxygen during severe muscular exercise and for the transfer of oxygen when alveolar P_{O_2} was low. This she did in her 1914 M.D. thesis, published in 1915.[4] She started with the assumption that "an essentially indifferent gas, like carbon monoxide, must pass through the alveolar epithelium by diffusion alone—an assumption which has never been questioned by anybody." She then improved her earlier technic[5] and described a single-breath test in which the subject first expired to residual air, then inspired deeply from a spirometer containing 1 per cent CO in air, then expired part way, held his breath for 6 seconds, and then completed the expiration. She sampled alveolar gas twice during the two-stage expiration (at the beginning and end of the breath-holding period) and measured the concentration of CO in each sample. From these and other measurements, she calculated the uptake of CO. It seems certain that Marie Krogh devised this first quantitative measurement of pulmonary diffusing capacity in man only to settle a controversy over a basic physiologic mechanism: does oxygen pass from alveolar gas to blood by secretion or only by diffusion? In her 1915 paper, she did measure diffusing capacity in 22 subjects and in 8 patients with asthma, emphysema, bronchitis, tuberculosis, or previous pneumonia, but apparently never used the test seriously for clinical diagnosis.

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The Kroghs clearly showed that two great physiologists were wrong in their theory of O_2 secretion. But even so, each of the two played an important role in developing Marie Krogh's CO test (in addition to provoking her to devise the test to prove conclusively that they were wrong!); each provided tools and knowledge that M. Krogh needed. Haldane had done important work on CO, had determined that hemoglobin had a very high affinity for CO compared to O_2, and that CO (apart from combining with hemoglobin) was a physiologically indifferent gas that did not enter into chemical reactions with tissues and could move through tissues only by diffusion. And Haldane (with Priestley) had developed a technic to sample alveolar gas, and M. Krogh had to obtain and analyze alveolar gas to calculate the amount of CO transferred during the 6-second breath-holding period.

As for Bohr—Lilienthal and Riley[6] credit him with laying the foundation for all methods that measure pulmonary diffusing capacity, whether for O_2 or for CO. Gas diffuses from alveoli to blood in pulmonary capillaries only when the partial pressure of the gas is higher in alveolar gas than that in blood. And the magnitude of the *difference* determines the rate of diffusion of gas; therefore, the rate slows when the PO_2 or PCO in alveolar gas decreases or when the PO_2 or PCO of pulmonary capillary blood increases. Since diffusing capacity is defined as the amount of gas transferred each minute for a difference of one-millimeter mercury in partial pressure across the membranes, it is essential that the pressure difference be measured accurately. Bohr[7] had developed the principles for calculating the *mean* partial pressure of O_2 in pulmonary capillary blood (which is *not* the average of the PO_2 in pulmonary artery blood entering the capillary and of the PO_2 in pulmonary vein blood leaving the capillary); Bohr's "integration technic" later (1946) the formed basis for Lilienthal and Riley's O_2 method.[8] But Bohr also recognized that, because of remarkable affinity of hemoglobin for CO, measurable quantities of CO could leave alveolar gas and combine with hemoglobin in capillary blood and all the while plasma PCO remained so low that it did not significantly decrease the difference between PCO in alveolar gas and in plasma and hence did not hinder the inward diffusion of CO; this formed the basis for M. Krogh's CO method because it permitted her to calculate CO uptake, without the need to sample mixed venous blood (impossible in 1914), and to measure its PCO.

August Krogh, in his article that disproved pulmonary secretion of O_2,[3] paid this tribute to Bohr, a year before Bohr died in 1911:

> I shall be obliged in the following pages to combat the views of my teacher Prof. Bohr on certain essential points and also to criticise a few of his experimental results. I wish here not only to acknowledge the debt of gratitude which I, personally, owe to him, but also to emphasize the fact, patent to everybody, who is familiar with the problems here discussed, that the real progress, made during the last twenty years in the knowledge of the processes in the lungs, is mainly due to his labours and to that refinement of methods, which he has introduced. The theory of the lung as a gland has justified its existence and done excellent service in bringing forward facts, which will survive any theoretical construction, which has been put or shall hereafter be put upon them.

Part two of this story began 35 years later. Except for a few isolated uses of Krogh's CO test by Barcroft[9] and Harrop and Heath,[10] her method was essentially forgotten or unused until 1950. In that year, I was asked to be Editor of a "methods volume" on Pulmonary Function Tests.[11] I designated one chapter "Gas-Blood Diffusion." Why, if Krogh's work had really been forgotten? Entirely because of a new approach by Lilienthal, Riley, and their associates at the Naval Air Station in Pensacola during World War II. In their work on aviation physiology, they had devised direct technics for measuring PO_2 and PCO_2 of blood and a new method for determining effective alveolar pressures of these gases. With these, they set out to determine whether an appreciable difference in PO_2 occurs between alveolar gas and arterial blood when man exercises at high altitude. From this research came their elegant 1946 paper[8] reporting on their brilliant, intellectually exciting use of physiologic principles to measure pulmonary diffusing capacity for O_2 (DL_{O_2}) in man.

So it was necessary in 1950 to have a "methods section" on Gas-Blood Diffusion and I prevailed upon Seymour Kety to write it. Some perhaps thought this was an inappropriate selection of author, because Kety was best known for his newly developed method for measuring cerebral blood flow in man—pretty far removed from the lungs. But Kety's method required that his subjects inhale nitrous oxide and he was in the process of writing a scholarly analysis of gas exchange at the lungs and other tissues for *Pharmacological Reviews*.[12] Kety studied all previous work on pulmonary diffusion of O_2 and CO and in his 1950 article[13] concluded:

> . . . the necessity for obtaining samples of mixed venous blood makes it appear doubtful that the determination of DO_2 by means of the Bohr equation will ever achieve clinical usefulness . . . [My note: Lilienthal and Riley's 1946 paper *assumed* values for PO_2 in mixed venous blood; the new technic of cardiac catheterization made it possible to obtain samples of such blood and replace assumed values with measured values.]
>
> Marie Krogh brilliantly side-stepped the difficulties inherent in the Bohr equation and measured the diffusion coefficient of

> the lungs by means of CO . . . It appears that the determination of D_{O_2} by Krogh's method [My note: by measuring D_{CO} and calculating D_{O_2} from it] would constitute a practicable and clinically useful technique for defining the diffusion characteristics of the alveolar membrane in health and disease.

That same year, Robert Forster joined our staff and at once set to work mastering the new infrared meter for rapid analysis of CO and our new mass spectrometer for rapid analysis of helium. Within a relatively short time, he, with Fowler and Bates, had modified Krogh's technic and developed a rapid and clinically useful single-breath technic.[14-16] In the same year, Filley independently reported on his steady state CO method.[17]

What have we learned from this account of the development of a clinical test? Certainly we've learned that eminent scientists can be wrong occasionally but that their being wrong sometimes provides the stimulus for an important discovery. We've learned that research on a fundamental physiologic problem can provide the basis for a practical clinical test. We've learned that putting good scientists into research laboratories in wartime can produce exciting basic advances and we've learned the value of asking a brilliant, disinterested scientist to be an "ombudsman" and indicate the directions for future work.

What we have *not* learned from this account is why there was a 35-year lag between Krogh's work and the development of a clinical test of D_{CO}, which, being non-invasive, did not have to wait for cardiac catheterization in man. It would be simple to say that pathology dominated clinical science until the 1940's and few clinicians had been trained to think physiologically. However, A. Krogh and Lindhard,[18] working in the same department as M. Krogh, had measured pulmonary blood flow (cardiac output) two years earlier using essentially the same noninvasive technic (measuring uptake of a soluble "inert" gas, nitrous oxide, as determined by its disappearance from alveolar gas)—and their test, modified in several ways, was used in the 1920s, 1930s, and 1940s until it was replaced by the direct Fick method.

It seems that something inherent in pulmonary medicine between 1915 and 1945 inhibited the development of tests of pulmonary function. Was it because pulmonary medicine in the first half of the twentieth century was really tuberculosis medicine that required largely roentgenograms and bacteriologic tests for diagnosis? All of us with a deep interest in pulmonary medicine should ponder on the reasons for the long quiet period in the early twentieth century because "Those who cannot remember the past are condemned to repeat it."

JULIUS H. COMROE, JR.

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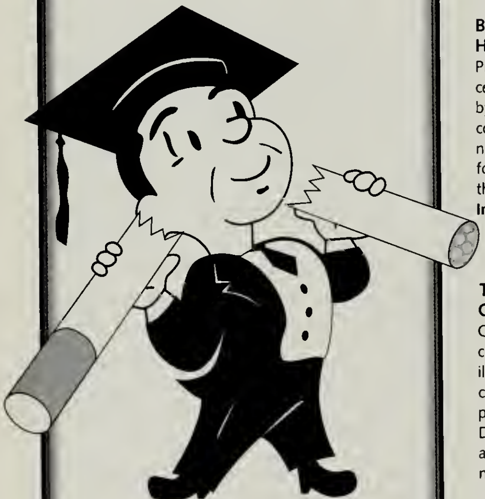

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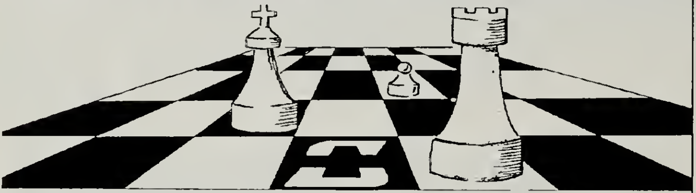

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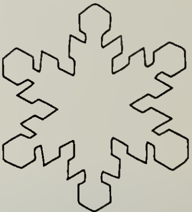

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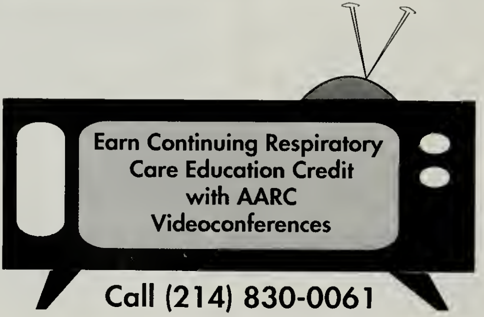

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The tape is intended for the would-be members of the discharge team and should be viewed when they suspect a patient might be a candidate for home ventilation. It can also be viewed by the prospective patient and his family. Key points that should be considered are highlighted: Are an adequate number of family members available to care for the patient? Are the necessary financial resources available? Does the home provide enough space and adequate electricity to manage the equipment? Do the patient and his family have a real understanding of his medical condition and prognosis?

The video also describes an 11-segment training program that should be completed by all caregivers prior to discharge. This program requires demonstrated competency in such areas as ventilator management and troubleshooting, suctioning, tracheostomy care, medication dispensing, infection control, and recordkeeping. The caregivers are required to spend a 24-hour 'live-in' period at the hospital during which they are responsible for all aspects of the patient just as they would be at home.

The script for this video was written by Jeffrey Lucas RRT, president of Northcoast Oxygen Company Inc, of Cleveland, Ohio. Mr Lucas's script is concise and demonstrates in a logical progression how the viewer can assemble the discharge team, work with the patient and family to give them realistic goals, and provide all of the needed clinical training for caregivers. The video does not instruct the viewer in specific recommended procedures such as suctioning protocols, ventilator care, or cleaning of equipment. It does, oddly enough, describe a procedure for tracheal stoma care, which includes a recipe for a peroxide-water solution and a recommended frequency.

The 17-minute video moves quickly and does not provide much detail on the 11 segments that the producers believe should be covered in the caregiver training; for example, it recommends ventilator troubleshooting be taught but does not provide any specific details about troubleshooting or what might cause an alarm. No information is provided regarding types of home medical equipment required or available or about financial issues such as insurance coverage or reimbursement guidelines. It is perhaps assumed by the producers that the viewer has sufficient knowledge of these areas; however, the person who understands these areas probably would have no need of the information on this tape.

There is no recommended time line to assist the viewer in understanding how long it may take to prepare a ventilator patient for discharge. From my own experience in working with ventilator patients in the home, I know that it is not uncommon for a hospital to expect to discharge a patient home with just a few days' preparation when in actuality it can take weeks or even months. Some guidelines would have been helpful to guide the team in setting a realistic, estimated discharge date based on all that must be accomplished.

This video can be a useful tool for the hospital that is just beginning to consider a patient for discharge. The likelihood of a safe and successful discharge can be improved by following its team approach, evaluating the patient, and instructing all caregivers as it recommends. Additional resources must be found for information regarding specific protocols and procedures, equipment, and financial issues that must be dealt with when working on a ventilator discharge.

Susan L McInturff RCP RRT
Clinical Director
Bay Area Home Health Care
Novato, California

Respiratory Care Principles: A Programmed Guide to Entry Level Practice, 3rd edition, by Thomas A Barnes EdD RRT in consultation with Jan Karel Schreuder MD and Jacob S Israel MD. Softcover, 515 pages, which includes tests, answer guides, and illustrations. Philadelphia: FA Davis Co, 1991. $39.95.

One of the first goals of all students enrolled in a respiratory therapy program is to achieve certification (CRTT) through the National Board for Respiratory Care. The principal criterion that many states use to award a license is a successful score on the Entry Level Exam. With so much to gain from a successful score on that exam, students will find this book valuable.

The genius of this book lies in its design. Material is presented in 242 instructional frames, which are divided into 19 chapters that include pretests, post-tests, illustrations, and a comprehensive exam in Chapter 17.

At the start of each chapter, a 20-question pre-test evaluates knowledge of the subject matter. Each chapter is then broken into frames, each frame a topic within the chapter that will give the student a solid overview without going into depth. At the finish of each frame, a multiple-choice question is provided to measure the reader's retention. The correct answer and explanations of incorrect choices are provided. At the end of each chapter, a 20-question post-test and workbook exercises help to improve the reader's skills and knowledge of the chapter covered.

Chapter 17 is a summative test of 200 questions that parallels the Entry Level Exam. By taking this test, the reader can measure his strengths and weaknesses and have time to review before taking the national review certification exam.

The book's format allows the reader to progress at his own pace, measure his retention, and, ideally, build confidence.

The author, Thomas A Barnes, is more than qualified to write such a credentialing guide book. Dr Barnes is Director of Clinical Education, Respiratory Therapy Program, and Assistant Professor of Respiratory Therapy, Department of Cardiopulmonary Sciences at Northeastern University. Past editions of the Guide have been used successfully by students and instructors of respiratory programs. This edition should be a welcomed addition to one's bookshelf and a key to a successful score on the Entry Level Exam.

Respiratory Care Principles is not limited to students in respiratory care but would be valuable to students of nursing, physical therapy, physician assistant programs, emergency paramedicals, and those enrolled in respiratory care correspondence courses. It could also be very helpful to instructors of respiratory care programs.

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Foundations of Respiratory Care, edited by David J Pierson MD and Robert M Kacmarek PhD RRT. Hardcover, 1306 pages, illustrated. New York: Churchill Livingstone Inc, 1992. $89.95.

Reviewing this book was a daunting task from the outset because of the sheer size of it (> 1,300 pages), and its scope. Because of its size, we divided the book roughly in half for reading purposes, with one of us (RRF) taking the first three sections and the other (JGS) the last two. We then discussed our reactions to the organization and concepts.

The book is divided into five large sections: Respiratory Structure and Function, Pathophysiology: How Disease Affects the Cardiorespiratory System, Assessment of Respiratory Function and Diagnostic Techniques, Clinical Approach to the Patient with Respiratory Disease, and Respiratory Care Practice. Within each of these sections are many generally short chapters, written by 1 of 56 contributors, each covering a small, discrete area of knowledge. This is both a strength and a weakness of the book. The focused nature of each chapter generally ensures that one will find nothing extraneous in the chapter—he will not have to wade through information that is not directly relevant to the topic. However, the small size of the 'bites' (apologies to television) causes two problems: (1) Material is sometimes somewhat artificially separated (for example, one chapter titled "Assessment of Lung Volumes" actually addresses only RV and the capacities containing RV, while the remainder of the volumes and capacities are covered in the next chapter titled "Assessment of Airflow"). (2) For complete coverage of a topic, one is sometimes directed to three or more other chapters in the book.

The first chapter covers, logically enough, the chemical and physical background, giving the clinician a basis for subsequent information. However, errors and confusing statements abound in Chapter 1—some merely editing problems, others factual in nature. The author, for example, asserts that inspired gas is at body temperature and is 100% saturated by the time it reaches the carina; an example on the same page gives Pb as 750 mm Hg, then uses 740 mm Hg in the actual calculation; a summary of the gas laws omits one subscript and gives another incorrectly; the author asserts that the relationship between pressure and volume in a normal lung is linear over the physiologic range; the inverse relationships between two quantities are referred to in every place except one as *indirect;* transmural pressure is defined as "the force exerted on the lateral wall of a system;" Bernoulli's Law states that "the *transmural* (italics ours) pressure is inversely related to the velocity of the gas;" a Reynolds number ≥ 2,000 means that turbulent flow definitely exists (rather than expressing a probability); the reader is directed to Table 1-5 for the ratios and gas flows for Venturi masks, but the proper table is 1-6; Table 1-6 adds 4 L/min source gas flow and 100 L/min entrained flow for a 24% mask and comes up with 100 L/min total flow; the Greek letter mu instead of etais used as the symbol for viscosity. The errors surface again in Chapter 5. In Chapter 5, The Thorax and Ventilatory Muscles: The Ventilatory Pump, the text states that the "transdiaphragmatic pressure(s) . . . positive at FRC and become (s) more positive during inspiration. . .," whereas Table 5-1 shows transdiaphragmatic pressure to be 0 at FRC and -10 at end inspiration; later in that chapter, Figure 5-7 indicates the serratus posterior *interior*. Figure 7-2 shows bronchioles starting at Generation 5, whereas Table 7-1 shows them starting at Generation 11. In Table 8-1, the units for resistance are given as dyne/s/cm^{-5}. Figure 9-1, a schematic depiction of the lung volumes and capacities, is described as the normal spirogram; in Figure 9-9A, there is a minus sign missing before 4, showing pressure in the airway during inspiration. Chapter 12 has several confusing parts. The author says that "CO_2 . . . dissolves in the plasma as P_{CO_2}." He also states that "The Cl^- that diffuses into the RBC associates with the K^+ released from the hemoglobin molecule . . .." (As a strong electrolyte, KCl is completely dissociated in solution.) A run-on sentence in the next paragraph is confusing until one (mentally) removes a comma and inserts a semicolon. At the end, the author again says that CO_2 is "carried as P_{CO_2}." In Chapter 13, the author states that "During depolarization, the intracellular flux of Na^+ cause the cell interior to become electrically *negative* (italics ours). In Chapter 20, Virazole is incorrectly spelled as is propranolol. Chapter 24 discusses the "patient" foramen ovale, Chapter 26, the motor end "place" of the muscle.

In two places (in the first half of the book), chapter authors redefine terms, an exercise which results in unnecessary confusion. In Chapter 21, the author defines FRC as "either the volume at which the static recoil forces of the lung and chest wall are balanced, or as that degree of end-expiratory lung inflation at which the work of breathing is least." This is problematic since the author refers to Chapter 9, in which FRC is defined solely as it is in the first half of this definition. In Chapter 30, the authors divide dead space into anatomic and alveolar or *physiologic* dead space; in Chapter 41, dead space is divided into anatomic and alveolar, with physiologic dead space being the sum of those two (the conventional definition).

In Chapter 27, the author states that "Another piece of evidence against pure alveolar hypoventilation during REM sleep is the relatively complete correction of desaturation by low-flow supplemental O_2." We believe that most textbooks state that the only pulmonary cause of hypoxemia that cannot be corrected by oxygen administration is pure intrapulmonary shunt. Finally, Chapter 42 shows its age (even having been published in 1992) when the author states that in management of childhood asthma, "inhaled adrenergic agents (serve) as the cornerstone." The author goes on to state that "Other chronic therapies may include oral theophylline and cromolyn sodium," while failing to mention steroids at all.

It gives us great pain to detail these problems with what has the potential to be a terrific book. We do so not to pick nits but to emphasize that there is not merely a rare typographic error, which was not caught, or a fact, which needs to be checked (as has occurred in every textbook ever published), but a multitude of them, especially in the early chapters. Although one can say that the errors can be corrected for the second edition, most of them should not have made it into the first edition.

Its shortcomings notwithstanding, the book makes use of a dazzling array of content matter experts, both physicians and therapists, in the field of respiratory care. The coverage is broad and of reasonable depth. In fact, one of us (JGS) found, for the first time, excellent coverage of a course content area. Case studies are generously and appropriately used; however, in the focused nature of the chapter, one is sometimes left wanting more. For example, in a case study (page 325) on the Adverse Pulmonary Effects on Cardiac Function (PEEP), one finds out that increasing the P_{aO_2} might not necessarily increase oxygen transport; the resolution of the dilemma is not provided, however.

Several chapters should be noted for their concise yet thorough coverage of a topic, combined with excellent readability. Chapter 44—Clinical Skills in Respiratory Care—spends more time than most on communication skills and professionalism; it also contains many 'nuggets' regarding the topographic landmarks for underlying structures. Chapter 47—Assessment of Gas Exchange and Acid-Base Balance—contains in fewer than 10 pages an excellent synopsis of acid-base dynamics that can stand on its own. Chapter 57—Diagnostic Imaging—contains a concise plan for reading radiographs, including a normal upright A-P film with *everything* labeled; it covers some of the most common pathologic conditions that one might see and also includes several conditions that mimic pneumothorax on the chest film.

Section 4 is a collection of 9 chapters that are, for the most part, clinically useful, practical, and current. This section and Section 5 will be of great interest to all practitioners, particularly educators who are looking for a "clinically oriented" approach to their courses, especially those courses that would be enhanced by a text that gives clinically useful information. Chapter 61—Cardinal Symptoms/Signs in Respiratory Disease—is a complete and logical presentation of this important aspect of assessment. Also quite useful is a table of "Most Likely Causes of Hemoptysis in Certain Patient Groups," which gives the clinician a wonderful and quick differential-diagnosis chart. Our only suggestion would be to add *graphic* representation of periodic breathing patterns. The next 5 chapters (62-66) are authored or co-authored by one of the editors (DJP). These are the "Clinical Approach" chapters dealing with common disorders and diseases of the cardiopulmonary system. These chapters are well-organized and 'user friendly' and thus should be very useful not only to educators and students but also practitioners because they contain practical dos and don'ts, approaches to management, algorithms and even a 10 Commandments approach to treatment of patients with acute-on-chronic ventilatory failure. In Chapter 62, Clinical Approach to the Patient with Obstructive Lung Disease, the author does an outstanding job in presenting the current treatment of asthma but neglects to mention the quite lengthy and well-documented information on asthma (as did Chapter 42's authors) published in June 1991 by the National Heart, Lung, and Blood Institute, "Executive Summary: Guidelines for the Diagnosis and Management of Asthma," Publication No. 91-3042A. Publisher's deadlines may account for this omission, and we must point out that the author is right on the mark in his clinical approach to assessment and management, which was very nearly the same as that detailed in the Executive Summary. Chapter 64 has a very good section on the ventilatory management of COPD patients—one of the greatest challenges to clinicians in respiratory care. This is one of the most concise and useful discussions on this topic we've seen to date.

Section 5, Respiratory Care Practice, contains 36 chapters that offer a comprehensive look at the modern practice of respiratory care. These chapters are chock full of generally useful and practical guidelines on the practice of the profession. Notably missing are references to the Clinical Practice Guidelines (incentive spirometry, pulse oximetry, oxygen therapy in the acute care hospital, spirometry, postural drainage therapy) published in this journal in December 1991. These guidelines would have enhanced the chapters on chest physical therapy (70), lung expansion therapy (73), assessment and management of acute atelectasis (74), and supplemental oxygen and other medical gas therapy (75). However, even with this omission, these chapters are an excellent reference for practitioners in respiratory care. As educators, we found this section particularly useful, with several of the chapters containing the answer to our prayers. We have been searching for a reference book that would give us and our students thorough, up-to-date, and clinically useful information on specific topics covered in our fall-semester, second-year course dealing with the clinical assessment and management of patients with cardiopulmonary disorders. Chapters 74, 81, 82, 86, 87, and 89-96 were immediately used to supplement this course, with great success. Chapters 76, 81, 82-87, and 101 will be extremely useful in our spring-semester course on mechanical ventilation. These chapters are well-written and easy to read—not something that can be said for most texts.

Once 'couthed up,' this book will be an excellent reference for any respiratory care practitioner's library. If we had not received this copy to review, we would have bought it anyway. With a $90 price tag, its use in an educational program would have to depend upon its "fit" with the courses and curriculum.

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Calendar of Events

AARC & AFFILIATES

February 16-19 in Reno, Nevada. The American Lung Association of Nevada and The Nevada Society for Respiratory Care present the 12th Annual High Sierra Critical Care Conference at the Peppermill Hotel Casino. Topics include adult, pediatric, and neonatal critical care issues. Contact Donna Turner, American Lung Association of Nevada, PO Box 7056, Reno NV 89510. (702) 829-5864 (9 AM-3 PM PST).

March 10-12 in Tahoe, Nevada. Chapter 9 of the CSRC and the American Lung Association of Alameda County return to South Shore Lake Tahoe and Caesar's for their 15th Annual Tahoe Conference. Topics to be covered include infectious diseases, transplantation, future team approaches, and high-tech in respiratory care. Also, a neonatal/pediatric brown bag lunch will be presented by Children's Hospital-Oakland. Contact Barbara Banks-Norton at (510) 275-8287 or Carrie Smith at (510) 893-5474.

April 26-28 in Philadelphia, Pennsylvania. The PSRC presents its 28th Annual Conference and Exhibition at the Adam's Mark Hotel. This year's theme is "Respiratory All-Stars: Building Pride Through Teamwork." The Pennsylvania State Sputum Bowl finals will be featured. Contact Betsy Schneck at (215) 829-3578 or Kathy Yandle at (215) 453-4517.

OTHER MEETINGS

February 11-14 in Orlando, Florida. The Florida Thoracic Society and the American Lung Association of Florida host the 15th Annual Pulmonary Wintercourse. Contact Marilyn O'Brien, ALA of Florida, PO Box 8127, Jacksonville FL 32239-0127. (904) 743-2933.

February 14-17 in Breckenridge, Colorado. The American College of Chest Physicians and St Helena Hospital present the Annual Meeting on "Cardiopulmonary Wellness and Rehabilitation." Contact Arlene Karavich at the American College of Chest Physicians (708) 498-1400.

March 3-5 in Lyon, France. The Journées Internationales de Ventilation à Domicile presents the International Conference on Home Mechanical Ventilation. The meeting includes scientific sessions, practical education sessions, and poster presentations and exhibits. Simultaneous translation (French/English) is offered. Deadline for abstracts is December 1992. Write: JIVD (SRMAR) 93, Grande Rue de la Croix-Rousse, 69317 Lyon Cedex 04, France. Call (33) 78 39 08 43, fax (33) 78 29 98 94.

March 11-13 in Washington, DC. The National Association of Medical Directors of Respiratory Care presents its 16th Annual Meeting at the Stouffer Mayflower Hotel. For more information, contact the NAMDRC Executive Office at 5454 Wisconsin Ave, Suite 1340, Chevy Chase MD 20815. (301) 718-2975.

March 26-28 in Nashville, Tennessee. The Association of Polysomnographic Technologists hosts the First Annual Spring Review Course in Advanced Polysomnographic Technology at the Opryland Hotel. Contact Todd Eiken, APT Education Chair, at (205) 592-5650.

March 24-27 in Helena, Montana. The American Lung Association of Montana presents the Twelfth Annual Big Sky Pulmonary Ski Conference. Contact the American Lung Association of Montana, 825 Helena Ave, Helena MT 59601. (406) 442-6556, fax: (406) 442-2346.

May 16-19 in San Francisco, California. The American Lung Association and American Thoracic Society host an International Conference. Vital information on the prevention and control of lung disease will be presented in a variety of symposia and workshops. Contact Maureen J O'Donnell, American Lung Association, 1740 Broadway, New York NY 10019-4374.

October 24-29 in Jerusalem, Israel. The XIVth World Congress of Asthmalogy convenes in Jerusalem. Topics presented include molecular biology in lung disease, cells involved in asthma, and house dust mite and asthma. Abstracts are currently being accepted. For more information, contact Gil-Kenes, 1617 JFK Blvd, Suite 946, Philadelphia PA 19103. (800) 223-3855.

1993 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Nashville, Tennessee, December 11-14, 1993. Accepted abstracts will be published in the November 1993 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications—READ CAREFULLY!

An abstract may report (1) an **original study,** (2) the **evaluation of a method or device,** or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/ rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. A **method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience *or* a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. A **case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form *and simple figures may be included provided they fit within the space allotted. No* figures, illustrations, or tables are to be attached to the abstract. Provide all author information requested in right column of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 7 (postmark). Authors will be notified of acceptance or rejection by *letter only*— to be mailed by August 15. Authors may choose to submit abstracts early. Abstracts received by March 19 will be reviewed and the authors notified by April 24. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 7).

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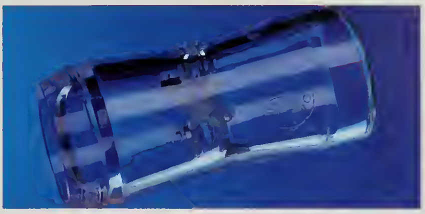

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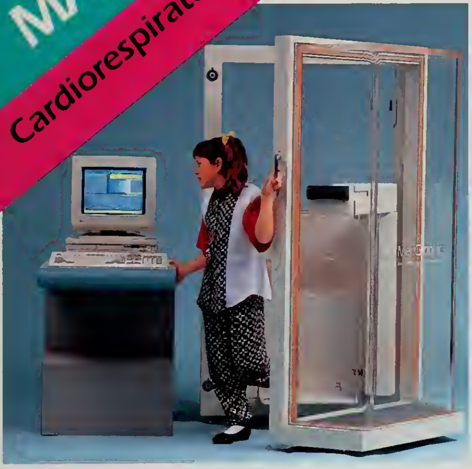

Our 1085/D Body Plethysmograph provides complete pulmonary function testing including: spirometry, maximal inspiratory/expiratory pressures, thoracic gas volume, and airways resistance using panting and non-panting techniques. Optional diffusing capacity (DLco) is also available (1085/DL).

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